Dysarthria In Early Parkinson's Disease

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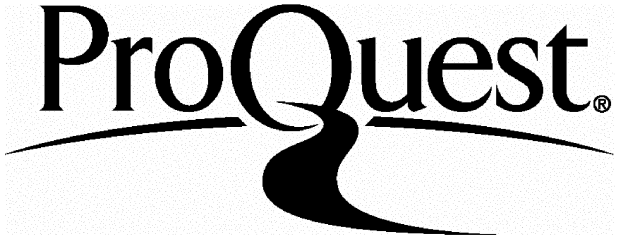

ABSTRACT

Dysarthria in early Parkinson's disease

The aim of the present study was threefold. First, to examine the incidence of dysarthria in patients in the beginning of Parkinson's disease by using a standardised test (Frenchay Dysarthria Assessment/FDA) and an intelligibility assessment tool. Second, to identify differences in speech and in measures of phonation between the Parkinsonian group and a matched control geriatric group using the FDA and electrolaryngography. Finally, to identify the effect of medication on speech and phonation in the dysarthric Parkinsonian group.

The results showed that 8 out of 12 (66%) Parkinsonian subjects exhibited lower scores in the FDA compared to controls. Qualitative differences between the two groups were found in the isolated movements of the articulators but not in running speech and speech intelligibility. An improvement in the FDA scoring was found 3-3.5 months after medication. This improvement focused on the areas of tongue and lips and was accompanied with significant increases in intelligibility. No differences in measures of phonation were found either between the two groups or in the same group after medication.

The above results suggest that in the beginning of Parkinson's disease, dysarthria is expressed as slowness and may be related to the primary diagnostic symptom of bradykinesia. Due to the small sample and the lack of dosage control, the significance of these findings appears to be inconclusive and warrants further investigation. Future research should employ instrumental quantitative measures on isolated movements of the articulators that may correlate with running speech and will aim to find clinical markers of speech in the diagnosis of Parkinson's disease.

To Elliana with special thanks

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CHAPTER 1. BACKGROUND TO THE RESEARCH QUESTION

Speech symptomatology in Parkinson's disease has been examined by numerous studies. The incidence of dysarthria has been reported to be variable, ranging from 3-89%. No study at the moment has examined the incidence of dysarthria immediately after the neurological diagnosis. The early inclusion of levodopa medication after the neurological diagnosis complicates the speech symptomatology in the disease. It is still unclear if dysarthric symptoms are improved, stay the same, or are worsened after the inclusion of medication.

This thesis examines the question of whether dysarthria exists in early-diagnosed Parkinson's disease and what its features are. The role of medication on speech and on selected voice characteristics is also examined. This chapter reviews the published literature on dysarthria in Parkinson's disease and frames the research questions.

1.1 Definitions and classification of Parkinsonism: Parkinson's disease

Parkinsonism is a group of neurological disorders marked by tremor at rest (present in the extremities and sometimes in the lips, chin, or tongue), rigidity, flexed posture in the entire body, bradykinesia-hypokinesia, loss of postural reflexes, and the freezing phenomenon. The diagnosis of definite Parkinsonism involves at least two of the above features with at least one being tremor at rest or bradykinesia (Fahn, 1995; Marjama-Lyons & Koller, 2001). It is generally accepted that decreased dopaminergic neurotransmission in the basal ganglia reflects the biochemical pathologies in Parkinsonism.

The classification of Parkinsonism is based on aetiology and includes four categories (Stacy & Jankovic, 1992):

- Primary or idiopathic Parkinsonism (Parkinson's disease, juvenile Parkinsonism)
- Secondary acquired or symptomatic Parkinsonism (due to infections, drugs, toxins, multi-infarcts, traumas, and others)
- Heredodegenerative Parkinsonism (Huntington's, Wilson's, and other diseases)
- Parkinsonism plus or multiple system degenerations (including diseases such as progressive supranuclear palsy, Shy-Drager syndrome, Alzheimer's disease, and others).

In the current study, emphasis will be given to idiopathic Parkinson's disease which involves more than 70%-76% of new cases of Parkinsonism (Hughes, Daniel, Kilford, & Lees, 1992; Roche, 1970; Stacy & Jankovic, 1992; Stern & Koller, 1993).

Parkinson's disease or idiopathic Parkinson's disease is a progressive neurological disease of unknown origin which is characterised by eosinophillic cytoplasmic inclusions (Lewy bodies) in the neurons and a degeneration of the pigmented neurons in the brain stem, substantia nigra (pars compacta), and locus coeruleus. At the onset of symptomatology, there is a loss of 60% of dopaminergic neurons and an 80% decrease of the dopamine content in the striatum (Fahn, 1995).

Clinically, Parkinson's disease is different to any other type of Parkinsonism (although this is a controversial issue among researchers). Its symptoms appear on only one side of the body, accompanied by an almost frequent rest tremor and it has a good response to levodopa treatment. If the patient does not respond well to levodopa, it is assumed that some other form of

Parkinsonism exists. So, levodopa treatment is not only a crucial factor in the alleviation of neurological symptomatology but also in the diagnosis of idiopathic Parkinson's disease.

1.1.1 Historical accounts of Parkinsonism and Parkinson's disease

James Parkinson (1817) made the first clear clinical description of Parkinsonism. At that time, Parkinson relied only on inspection and observation to describe the disease that was named "shaking palsy" or "paralysis agitans". His contribution to the recognition of the disease was remarkable. He was the first to describe the disease as an entire entity, to distinguish its tremor from other tremors that were caused by alcoholism, senility, or caffeinism and his work led to further anatomical examination of Parkinsonism.

Before Parkinson many medical writers focused on the varieties of tremor (resting vs. action tremor) including Galen (200 AD) who however attributed the tremor to chronic alcoholism (Roche, 1970). Calne, Dubini, and Stern (1989) included Leonardo Da Vinci among the early contributors to the identification of the disease by his observations and associations of the problems of voluntary movement with the tremor. Parkinson (1817) stated that Hippocrates, Franciscus (Sylvious) de la Boe (1617-1672), and Boissier de Sauvages (1768) were early contributors in the identification of the disease signs. The first seemed to mention tremor in his scripts, the second separated clearly the resting tremor from tremor during a voluntary act and the latter described the characteristic gait of Parkinsonism.

In the early 19th century Charcot and Vulpian (1861-1862) proposed the name "Parkinson's disease" and noted tremor as the distinguishing symptom

(together with memory and intellectual problems) during the late stages of the disease. After Charcot and his colleagues had placed a special emphasis on Parkinson's disease, many authors devoted themselves to the description of the disease. Among them was Gowers (1893), who summarised and expanded the symptomatology of the disease in his personal notes of 80 cases. Tyler (1992) reports that the author who crystallised the modern conception of paralysis agitans was Gowers. It seems that Gowers was also the first who referred to the monotony of speech in Parkinson's disease commenting on the delay in the beginning of the sentence and on the rapidity of the following uttered words.

1.1.2 Epidemiological factors of idiopathic Parkinson's disease

Epidemiological studies suggest a worldwide appearance of the disease with a variable range of about 100-200 cases per 100,000 people (Marttila, 1992; Tanner & Ben-Shlomo, 1999). A concomitant variation of Parkinson's disease is found in the incidence rates with a range from 5-24/100,000. In the UK and US the incidence rises about 1-2% in people after the age of 50 (Marjama-Lyons & Koller, 2001; Mutch, Strudwick, Roy, & Downie, 1986). No difference between the sexes or a slight male preponderance has been found (Marttila & Rinne 1976; Rajput, Offord, Beard, & Kurland, 1984; Tanner & Ben-Shlomo, 1999). The epidemiology of Parkinson's disease reflects a variability in the prevalence and incidence rates because of diagnostic difficulties in early recognition, a differential diagnosis of other tremors and extrapyramidal syndromes (Marttila, 1992) and/or differences in study design (Tanner & Ben-Shlomo, 1999).

Aetiologically, many risk factors for developing Parkinson's disease have been discussed. Tanner (1992) summarised eight risk factors that include: age,

gender, race, genetic predisposition, toxicant exposure, trauma, and emotional stress. Age is an important risk factor of the disease. The current picture shows scarcity of the disease before the age of 30, a low incidence between 30 and 40 years, a sharp increase in incidence thereafter with a peak between 70 to 79 years and a declination of incidence for higher ages (Baldereschi et al., 2000; Marttila, 1992; Tanner, 1992; Tanner & Ben-Shlomo, 1999). Other authors report the peak of incidence in the ages between 75-84 years (Rajput et al., 1984).

As already noted, some reports show no gender differences in prevalence rates (Shastry, 2001; Tanner, 1992) or slight male preponderance (Tanner & Ben-Shlomo, 1999). Exceptions to these findings were two studies, one study in China (Li et al., 1985) which found that men were 3.7 times more likely to develop Parkinson's disease than women, and, another study in Italy (Baldereschi et al., 2000) which found that men were twice as likely to develop Parkinson's disease than women.

The Caucasian population has been reported to be at a greater risk compared to the other races, even though no conclusions have been extracted about the geographical pattern of the disease from this evidence (Marttila, 1983; Tanner, 1992). A lower occurrence of the disease is found in the African and Asian races compared to Caucasians who seem to have the highest prevalence rate of Parkinson's disease. Exceptions to the above reports are the results from the studies of the Parsi colony in Bombay (India) and Copiah county in Mississippi (U.S). The first showed similar prevalence to that found in Europe and North America, while the second showed a similar prevalence rate between whites and blacks (Bharucha, Bharucha, Bharucha, Bhise, & Schoenberg, 1988; Schoenberg, Anderson, & Haerer, 1985).

While studies in the earlier decades focused on the influence of environment, after 1990 there has been a focus on genetics to explain idiopathic Parkinson's disease. The evidence shows that genetic factors are involved in early and late-onset Parkinson's disease. Early-onset Parkinson's disease refers to autosomal recessive forms of Parkinson's disease while late onset Parkinson's disease refers to the idiopathic form of Parkinson's disease. The Parkin gene was found to contribute to the development of early-onset Parkinson's disease while several genes were found to contribute to the development of late-onset Parkinson's disease (Scott et al., 2001).

Other studies associate different types of exposures such as rural residence, farming, well-water drinking, smoking, or herbicide/pesticide with Parkinson's disease (Koller et al., 1990; Tanner et al., 1989; Wong, Gray, Hassanein, & Koller, 1991). Recent evidence suggests that among other factors there is an association of the existence of domestic animals at home and Parkinson's disease, with the Parkinsonian subjects (especially before the age of 20) reported as having fewer domestic animals and lower duration of animal contacts. The appearance of domestic animals was suggested to have a protecting effect in the development of Parkinson's disease (Kuopio, Marttila, Helenius, & Rinne, 1999). In this study, even though no negative association between smoking and Parkinson's disease was found, the increased mean age of onset for Parkinson's disease in ever-smoking subjects as compared to the never-smoking subjects possibly suggest that smoking delays the onset of Parkinson's disease. In general, the picture of an infectious environmental agent and Parkinson's disease is still not clear and further investigation is needed. It is unlikely that a sole etiologic environmental agent exists especially when it should

be present for almost a century from the time that James Parkinson discovered this disease (Duvoisin, 1999).

Head injury also appears in the literature to be associated with Parkinson's disease (Bharucha et al., 1986; Tanner et al., 1987). However, recent evidence does not support this association (Kuopio et al., 1999). Other studies support the notion that the association of head trauma and Parkinson's disease may be caused by a biased recall on the part of the patients (Tanner, 1992; Tanner & Ben-Shlomo, 1999). Some authors include the effects of boxing on brain damage as a result of a syndrome called "punch drunk" that mimics some of the Parkinsonian features such as the shuffling gait and the dysarthria (Corsellis, Bruton, & Freeman-Browne, 1973; Corsellis, 1989). Although it is not recognised officially that boxing results in this syndrome, damage to the motor pathways in the cerebellum and the substantia nigra is reported. The clinical features of this pathological state include dysarthria with and without equilibrium and spasticity, or, rigidity and striatal tremor associated with dementia. As in Parkinson's disease, the lack of large pigmented neurons -among other features- in the substantia nigra is considered a histological finding of this syndrome.

Emotional stress has also been associated with the development of Parkinson's disease, but more research is needed to give a clear picture of this risk factor. Duvoisin (1999) reports that age, gender, and stress may influence the exact moment of appearance of the disease and its clinical symptomatology.

Additional risk factors have been discussed by Marttila and Rinne (1986) including other diseases (cancer, occurrence of stroke, coronary heart disease, and common viral infections), essential tremor and specific diet, but no associations have been found with Parkinson's disease. In contrast to the above

findings, Tanner and Ben-Shlomo (1999) report that there is an increased possibility of diet as a possible exposure factor. Evidence from a cohort study of Far East prisoners of war who experienced dietary insufficiency between 1942-1945 showed an increased prevalence rate of 512 cases / 100,000 persons (24 people out of 4,684 subjects) far beyond the usual prevalence rate of 100-200 cases / 100,000 people. Finally, other studies have found associations of Parkinson's disease and increased exposure to mercury (Ngim & Devathasan, 1989) or increased exposure to manganese, iron, and aluminium (Zayed et al., 1990).

The time of exposure to an environmental factor in relation to the cause of Parkinson's disease has also been discussed (Marttila & Rinne, 1986). It has been suggested that there might be an exposure throughout the life of persons who develop Parkinson's disease that has a low frequency during childhood and after 60 years of age and a maximum occurrence between 30 to 40 years. Tanner and Ben-Shlomo (1999) state that a long latency period of around 20-30 years should exist as a time of exposure. This period is probably followed by a period in which there is a reduction of the number of substantia nigra neurons by 70-80% and the reduction of striatal dopamine. Both of them were found long before the appearance of the clinical motor symptoms (Marsden, 1982; Tanner & Ben-Shlomo, 1999).

Marttila and Rinne (1986) support the notion that the incidence and prevalence rates of Parkinson's disease have been stable many decades and so modern technology might not be the cause of the disease. Also, the specific environment (town, village) of the person prone to the disease, might be responsible for the disease but not the environment at the family level. Finally,

the appearance of infections and toxins especially in postencephalitic Parkinsonism and MPTP-induced Parkinsonism that mimic the symptoms of Parkinson's disease might be serious candidates among others for the development of the disease. What is uncertain at the moment is "the exact timing of the process leading to Parkinson's disease...and the nature of the cause of this disease" (Marttila, 1992, p. 50). In contrast, other researchers argue that the existence of syndromes similar to Parkinson's disease due to the effect of environmental agents does not necessarily prove their direct association to Parkinson's disease. Furthermore, no agent seems to reproduce the same neuropathology of Parkinson's disease (Duvoisin, 1999).

1.1.3 Neuropathology and aetiology in idiopathic Parkinson's disease

It is well known and has been well established by researchers in the 20th century that Parkinson's disease arises as a result of basal ganglia and more specifically substantia nigra pathology (Hornykiewisz, 1966; Wilson, 1912). Gibb (1992) summarised the neuropathology of Parkinson's disease. According to him, there is a moderate to severe loss of cells in the zona compacta of the substantia nigra. At the same time the remaining cells include eosinophillic Lewy bodies in great quantity especially in the neurons with long axons. If there is no existence of Lewy bodies, the disease is excluded from the neurological diagnosis. Another type of cell inclusions that is found in Parkinson's disease (substantia nigra and locus coeruleus) is the pale body. As the name suggests the loss of melanin pigment in the substantia nigra results in the characteristic pale colour.

The existence of Lewy bodies at an early stage is associated with the

degeneration of nerve cells. Often Lewy bodies are found before any significant cell loss. Their distribution shows the sites of degeneration, namely, the substantia nigra, locus coeruleus, ventral tegmental area, nucleus basalis of Meynert, thalamus, the entire autonomic nervous system, raphe nuclei, and cerebral cortex. The cause of the degeneration of dopamine-containing cells and the existence of Lewy bodies in the zona compacta of the substantia nigra is still unknown (Jenner, 1992).

Even though in the past, the majority of studies that searched for aetiologic factors of Parkinson's disease focused on possible environmental risk factors, recently this view has changed. The current view is that there might be "an interaction of genetic and environmental risk factors, in which specific genetic templates are more susceptible to the influences of environmental exposures" (Scott et al., 2001, p. 2243). More specifically, a deficiency of a gene product might be the trigger for the toxic action of an environmental factor to produce Parkinson's disease (Duvoisin, 1999). Despite this interplay between genetics and environment, gene therapy in the future might be able to treat Parkinson's disease (Shastry, 2001).

In summary, the neuropathological process of Parkinson's disease is the result of the selective degeneration of the dopaminergic nigrostriatal pathway, which results in reduced striatal dopamine concentrations. The signs and symptoms of the disease (tremor, rigidity, and bradykinesia) emerge as a consequence of the above process. Further explanatory mechanisms of the lack of dopamine will be discussed in section 1.9.2. The current aetiological view is that Parkinson's disease is the product of the interplay between genetic susceptibility and the toxic action of one or more environmental factors.

1.2 Incidence of dysarthria in Parkinson's disease

Dysarthria is defined as "...a collective name for a group of speech disorders resulting from disturbances in muscular control over the speech mechanism due to damage of the central or peripheral nervous system. It designates problems in oral communication due to paralysis, weakness, or incoordination of the speech musculature" (Darley, Aronson, & Brown, 1969a, p. 246). Hypokinetic dysarthria is a motor speech disorder associated with lesions in the basal ganglia control circuit. It is primarily the result of Parkinson's disease.

According to Darley et al. (1969a), the speech characteristics of hypokinetic dysarthria are (from most severe to least severe): monopitch, reduced stress, monoloudness, imprecise consonants, inappropriate silences, short rushes of speech, harsh voice quality, breathy voice (continuous), low pitch, and variable rate. Prosodic insufficiency is considered a cluster of abnormal speech characteristics in hypokinetic dysarthria as compared to other dysarthria types (Darley, Aronson, & Brown, 1969b). It includes monopitch, monoloudness, reduced stress, short phrases, variable rate, short rushes of speech, and imprecise consonants. Darley, Aronson, and Brown (1975) state that "the association of perceived acoustic characteristics most distinctive of hypokinetic dysarthria comprises significantly reduced variability in pitch and loudness, reduced loudness level overall, and decreased use of all vocal parameters for achieving stress and emphasis" (p. 195). All the other speech characteristics such as imprecise consonants, harsh and breathy voice quality occur at variable rates. More details about these studies will be given in section 1.5.1.

The speech characteristics of hypokinetic dysarthria are thought to be due to the reduced range of movement that is caused by the rigidity of muscles. This rigidity involves the laryngeal and the vocal tract musculature (Kent, 1990) as well as the respiratory musculature (Critchley, 1981). The consequences of rigidity might involve bowing of the vocal folds, fast rate, reduced movements, and inadequate oral closures.

The incidence of hypokinetic dysarthria in Parkinson's disease has been found to be variable in different studies. A detailed overview of these studies will follow. A high incidence was found by Logemann, Fisher, Boshes, and Blonsky (1978). The authors examined 200 patients with Parkinson's disease and found that 89% of them exhibited phonatory problems (breathiness, roughness, hoarseness, tremulousness), 45% exhibited articulatory problems, 20% exhibited rate abnormalities (short or long pauses, syllable repetition, and abnormally long syllables) and 10% of them exhibited hypernasality.

In a follow-up study, 227 subjects with Parkinsonism (183 subjects with idiopathic Parkinson's disease and 44 subjects with postencephalitic Parkinsonism) exhibited variable incidence rates (Hoehn & Yahr, 1967). Seven subjects with idiopathic Parkinson's disease (3%) and 12 subjects with postencephalitic Parkinsonism (27%) reported speech disturbances. However, no information was given in this study for medication status (this study coincided with the advent of levodopa, so probably no medication was given to patients) and speech was recorded as one of many other symptoms of the disease.

Scott, Caird, and Williams (1985) reported that, in general, speech disorders in Parkinson's disease occur in half of all cases. Mutch et al. (1986) assessed 265 patients with idiopathic Parkinson's disease (median duration of

the disease 7.2 years) using two rating systems: the Webster scale and the Hoehn and Yahr rating system. The results, among others, showed that 64.9% of patients who were assessed with the Hoehn and Yahr rating system exhibited some speech difficulty and 71.5% of patients who were assessed with the Webster rating exhibited speech problems of varying severity: mild (37.4%), moderate (25.7%), and severe (8.4%).

Hartelius and Svensson (1994) found that 70% of Parkinsonian patients perceived that their speech and voice were impaired (duration of the disease from 3-10 years). More specifically, sixty one percent (61%) of the total number of the Parkinsonian patients (N = 195) reported weak voice, 36% reported imprecise articulation, 32% reported hoarseness, 27% reported difficulties getting started, and 17% reported monotonous voice. A strong positive correlation was found between the duration of the disease and the severity of speech symptomatology.

More recently, Coates and Bakheit (1997) found that 64.6% of 48 Parkinsonian patients (mean duration of disease 6.7 years) exhibited speech problems (reduced intelligibility of speech). Fifty percent (50%) of them displayed mild speech disturbances and the remaining 14.6%, exhibited moderately severe or severe dysarthria. One third of the subjects were found to be unaware of their speech problems. Contrary to the study by Hartelius and Svensson (1994), this study found a poor correlation between the severity of Parkinson's disease and reduced intelligibility of speech and a weak negative correlation between intelligibility of speech and duration of disease.

From the above findings, it seems that the sampling of the population of Parkinsonian patients occurred at different times during the course of the

disease. In addition, the incidence rates of some studies were based on history forms (Hoehn & Yahr, 1967), questionnaires (Hartelius & Svensson, 1994) or speech assessment based on neurological scales alone or with intelligibility assessment (Coates & Bakheit, 1997; Mutch et al., 1986). The variable findings of these incidence rates may also be related "to the progressive nature of the disease and/or to the individual responses to anti-Parkinsonian medication" (Theodoros & Murdoch, 1998, p. 271). In order to exclude factors such as duration of disease and medication, it is suggested that new research may need to examine the dysarthria of patients with Parkinson's disease immediately after the neurological evaluation and diagnosis. In this way, medication will not have had chance to affect speech and a clearer picture of dysarthria as a primary symptom of idiopathic Parkinson's disease will arise.

1.3 Cognitive impairment in Parkinson's disease

This section aims to summarise how cognitive ability is affected by Parkinson's disease. It is imperative in the present study that the examination of dysarthria will exclude factors such as cognitive impairment. The reported cognitive problems in Parkinson's disease involve the appearance of dementia. Dementia is defined as a disorder that exhibits impairment in short term and long term memory and abnormalities in at least one of the areas of mental function: abstract thinking, judgement, language, praxis, visual recognition, constructional ability, or personality (American Psychiatric Association, 1987). Even though this definition is applied basically to the dementia of Alzheimer's disease, it is not clear if the same criteria could be used in idiopathic Parkinson's disease (Sagar, 1992) due to probable behavioural differences between the two diseases. In

general terms, dementia is defined with the appearance of two out of four cognitive domains (language, memory, executive function, and visuospatial function) (Piatt, Fields, Paolo, Koller, & Tröster, 1999).

The prevalence of dementia in Parkinson's disease is approximately 20-43% increasing with the duration of the disease (Aarsland et al., 2001; Cummings, 1988; Levy et al., 2002; Sagar, 1992). The risk of developing dementia has been investigated in cohort studies (Aarsland et al., 2001; Levy et al., 2002). Patients with Parkinson's disease had a 6 times higher risk of dementia than general elderly people (Aarsland et al., 2001). Age, duration of disease, Mini Mental State Examination score, Hoehn and Yahr stage (>2), and levodopa dose were found to be predictors of dementia in the Parkinsonian group when the Parkinsonian patients were re-evaluated after 4 years (Aarsland et al., 2001). Levy et al. (2002) found that the combined effect of age and severity of extrapyramidal signs was a risk factor of developing dementia. Another study found that speech and bradykinesia were associated with incident dementia (Levy et al., 2000). No information about duration of the disease was given in this study. Also, dysphagia and Parkinson's disease (with and without dementia) co-occur. In fact, one quarter of Parkinsonian subjects with dementia and one-half of the Parkinsonian subjects without dementia are documented to also have dysphagia (Bine, Frank, & McDale, 1995).

The dementia of Parkinson's disease is often called subcortical dementia. Subcortical dementia is a syndrome that is characterised by dysarthria, memory impairments, executive deficits, depression, and prominent motor disability compared to dementia of Alzheimer's disease. In contrast, dementia of Alzheimer's disease is characterised by aphasia, long-term memory deficits,

agnosia, normal mood, and minor motor disability (Sagar, 1992). However, there are researchers who oppose the distinction between cortical and subcortical dementia (Mayeux, Stern, Rosen, & Benson, 1983). The concept of subcortical dementia has been attributed to the effect of dopamine deficiency on intellectual function because:

- Dementia improves for the first 1-2 years of therapy with levodopa.
- Its severity has been correlated with dopamine loss and akinesia.
- It has been associated with other hypodopaminergic states such as supranuclear palsy and MPTP-induced Parkinsonism (Cummings, 1988).

More specifically, Cummings and Benson (1984) described and distinguished subcortical dementia compared to cortical dementia in different levels (severity, personality, motor system involvement, anatomy, and neurotransmitter involvement). In severity, memory and cognition are more impaired in cortical dementia rather than in subcortical dementia. In personality, cortical dementia is euphoric as compared to the subcortical dementia that is apathetic. In motor system involvement, the subcortical dementia presents with abnormalities in posture, gait, motor speed, movement, and speech (dysarthria). In anatomy, subcortical dementia involves the basal ganglia, the thalamus, and the mesenchephalon. Finally, in neurotransmitter involvement, subcortical dementia presents with a lack of dopamine in contrast to the cortical dementia of Alzheimer's disease, which presents with a lack of acetylcholine.

Some studies aimed to prove differences between cortical vs. subcortical dementia by measuring capacities such as memory, visuospatial function, mental function, and language (Cummings, Darkins, Mendez, Hill, & Benson, 1988; Goldman, Baty, Buckles, Sahrmann, & Morris, 1998; Huber, Shuttleworth,

& Paulson, 1986). All studies assessed dementia using neuropsychological test batteries. Huber et al. (1986) found that dementia in Parkinson's disease was characterised by mild impairment of memory and visuospatial function but not impairment of language or evidence of apraxia. Cummings et al. (1988) found that the Parkinsonian patients exhibited motor speech, writing, and language abnormalities (phrase length and grammatical complexity) compared to patients with Alzheimer's disease. Parkinsonian patients with and without dementia showed more speech and writing abnormalities than the patients with Alzheimer's disease and dementia. Murray (2000) examined differences in language between two groups, one with Huntington's disease and one with Parkinson's disease. The results showed that the subjects with Huntington's disease exhibited shorter and less syntactically complex utterances (subject-verb-object utterances) when describing the Cookie-Theft picture. The author concluded that the neural structures that affected by Huntington's disease (e.g., caudate nucleus, putamen) contribute more to syntax production than these of Parkinson's disease (e.g., substantia nigra).

The manifestations of subcortical dementia compared to non dementia and healthy elderly people were also examined. Goldman et al. (1998) found a lower performance of Parkinsonian subjects with dementia compared to non demented Parkinsonian subjects and controls in logical memory (recalling of stories) block design (arrangement of blocks to match designs), digit symbol (transcription of numbers and symbols), and trailmaking (connection of numbers with lines). Piatt et al. (1999) found that demented Parkinsonian subjects scored more poorly in semantic, phonemic, and action fluency tasks compared to controls and Parkinsonian non demented subjects. According to the authors, the

action fluency task is probably an early indicator of the conversion from Parkinson's disease without dementia to Parkinson's disease with associated dementia. This lateral finding agrees with other reports that action fluency was a more efficient task in the identification of Parkinson's disease without dementia and Parkinson's disease with dementia (Woods, Tröster, & Fields, 2001).

Other studies examined grammar and syntax in Parkinsonian demented patients (Lieberman et al., 1992; Small, Lyons, & Kemper, 1997). The results of these studies show that grammatical ability is associated with the severity of dementia in Parkinson's disease whereas syntactic comprehension is associated with the neurological stage of Parkinson's disease and the complexity of the sentence.

Small et al. (1997) investigated the grammatical abilities of patients with mild and moderate dementia and Parkinson's disease, patients with Parkinson's disease and no dementia, and controls, by comparing written language samples of single sentences. The results showed that impairments in grammatical ability vary with severity of dementia. Thus, the Parkinsonian patients with mild dementia and no dementia exhibited normal grammatical production as compared to patients with moderate dementia who wrote shorter sentences with a reduced propositional density. These findings when compared to the findings by Cummings et al. (1988) and Illes (1989) suggest that the dementia of Parkinson's disease affects the lexical-semantic content more than the syntactic content of grammatical production (Small et al., 1997).

1.3.1 Cognitive impairment in early Parkinson's disease

Although studies of cognitive impairments in Parkinson's disease have

given variable results due to a possible mixture of subjects with variable duration of disease, less research has taken place with patients with early Parkinson's disease. Levin and Katzen (1995) define early Parkinson's disease to occur in less than five years duration, or for the severity of symptoms to be at stages I and II in the Hoehn and Yahr scale. Even though there is an apparent effort to exclude factors such as medication and duration of disease in order to study early Parkinson's disease, there are still differences between studies. Duration of disease is the most apparent one. Even though the mean duration of disease does not seem to change across studies (21-28 months), the range of this duration is different. Other studies exhibit a range of 3-96 months (Farina et al., 2000), other 6-36 months (Levin, Llabre, & Weiner, 1989), and others to 3-48 months (Cooper, Sagar, Jordan, Harvey, & Sullivan, 1991; Lees & Smith, 1983).

In summary, a number of studies report the general preservation of naming and fluency (phonemic and semantic) as manifestations of language in early Parkinson's disease (Farina et al., 2000; Lees & Smith, 1983; Levin et al., 1989; Levin & Katzen, 1995). Two studies found some differences in phonemic fluency (Lees & Smith, 1983) and semantic fluency (Cooper et al., 1991) but these were not generalised (only one out of three letters in words that produced in one minute and only the semantic category of objects showed a significant difference).

Set-shifting (shift between different mental sets) has been reported to be consistently impaired in subjects with early Parkinson's disease (Cooper et al., 1991; Farina et al., 2000; Lees & Smith, 1983; Levin et al., 1989; Levin & Katzen, 1995). The Parkinsonian subjects tended to make more errors when they were required to shift from one mental category to another. Farina et al.

conclude that set shifting and maintaining is the first executive ability to be lost in the early stages of Parkinson's disease. Kanazawa, Hirotaro, and Yoshikuni (2001) reported that executive function impairments coincide with the stage of Parkinson's disease (Hoehn and Yahr II-III neurological stages) in which the bilateral symptoms start to show.

Immediate and delayed memory has been reported to be impaired in different studies (Cooper et al., 1991; Farina et al., 2000; Levin et al., 1989). The role of impairment of memory in early Parkinson's disease is not so clear due to the effect of medication that may disrupt memory skills (Levin & Katzen, 1995). The authors support the notion that further research is needed to investigate if memory impairment is a marker of Parkinson's disease or a precursor of dementia in Parkinson's disease. Along the same lines, Cooper, Sagar, and Sullivan (1993) attributed the impairment of memory in the early Parkinsonian subjects to their deficits in attention and not to a generalized impairment of cognition.

1.4. Diagnostic criteria for Parkinson's disease and the use of medication

The neurological diagnosis of Parkinson's disease is based on the clinical examination. At the moment there is no physiological test to diagnose this disease. It is well known that the clinical diagnostic criteria for Parkinson's disease do not exclude the possibility of a misdiagnosis. The clinical heterogeneity that is manifested in the Parkinsonian population further complicates the diagnosis (Koller, 1992). A broad approach involving not only inclusion but also exclusion criteria was suggested as a solution to the problem of accurate diagnosis of Parkinson's disease (Koller & Montgomery, 1997).

Most of the researchers agree that the inclusion criteria should involve the presence of two of the three cardinal features of Parkinsonism:

- Bradykinesia
- Tremor
- Rigidity

Some researchers include postural instability in the aforementioned criteria (Calne, Snow, & Lee, 1992; Gibb & Lees, 1989; Hermanowicz, 2001; Quinn, 1997). Specific diagnostic criteria have been established based on the results of the Parkinson's Disease Society Brain Bank (PDSBB) which receives brain tissue from all over UK and examines it histologically. These criteria increased the accurate diagnosis of Parkinson's disease to 82% (Hughes et al., 1992). Hughes et al. lists all the PDSBB clinical diagnostic criteria (inclusion and exclusion) for Parkinson's disease (Figure 1).

1) Bradykinesia plus one of: rigidity, tremor, and postural instability.
2) Exclusion criteria: history of repeated strokes, history of repeated head injury, history of encephalitis, oculogyric crises, neuroleptic drugs at onset of symptoms, more than one Affected relative, sustained remission, strictly unilateral features after 3 years, supranuclear Gaze palsy, cerebellar signs, early severe autonomic involvement, early severe dementia With disturbances of memory, language and praxis, Babinski sign, presence of cerebral Tumour, negative response to large doses of levodopa, MPTP exposure.
3) Three or more of the following supportive positive criteria: unilateral onset, resting tremor present, progressive deterioration, persistent asymmetry affecting the side of onset, Excellent response to levodopa (70-100%) initially, severe levodopa-induced chorea, Response to levodopa for 5 or more years, duration of disease of 10 years or more.

Figure 1. UK Parkinson's Disease Society Brain Bank clinical diagnostic criteria.

Bradykinesia (slowness of movement) and tremor followed by rigidity are the most frequent features of Parkinson's disease. Terms such as akinesia (paucity of movement), hypokinesia (reduced range of movement), and bradykinesia have been used interchangeably to describe the loss of the ability to move (Marsden, 1989). The absence of facial expression in Parkinsonian patients is an expression of akinesia while limb slowness during movement is an expression of bradykinesia. Finger tapping is a task that is used by neurologists to observe the existence of bradykinesia. Tremor is usually presented as a unilateral hand tremor, more noticeable at rest and decreased when the affected hand is used. Finally, rigidity (stiffness of movement) is manifested as an increased tone throughout the range of limb movement. While bradykinesia and tremor are usually reported by patients, rigidity is not (Hermanowicz, 2001).

The development and use of levodopa (L-DOPA) almost 25 years ago revolutionised the treatment approach for Parkinson's disease. Levodopa restores the low cerebral dopamine levels in Parkinson's disease. It is still the main medication for idiopathic Parkinson's disease. However, as the disease progresses motor fluctuations and dyskinesias occur after 3-5 years of levodopa medication. For this reason, there are alternative treatment strategies that hope to slow or halt the progression of Parkinson's disease. Some of them are proposed to be complementary to levodopa while others are used as early monotherapy. All the different medications for Parkinson's disease tend to alleviate the mobility symptoms especially in large limb and body movements (Hermanowicz, 2001). Figure 2 lists all commonly described drugs for Parkinson's disease in Europe and US.

ACTION	BRAND NAME	GENERIC NAME
Anticholinergics	Artane	Trihexyphenidyl
	Tremen	Trihexyphenidyl
	Kemadrin	Procyclidine
	Akineton	Biperiden
	Cogentin	Benztropine
	Symmetrel	Amantadine
	Parsidol	Ethopropazine
Dopamine receptor agonists	Parlodel	Bromocriptine
	Permax	Pergolide
	Dopergine	Lisuride
	Requip	Topinirole hydrochloride
	Mirapex	Pramipexole
Levodopa-decarboxylase	Sinemet	Carbidopa/levodopa
Inhibitor combinations	Sinemet CR	Carbidopa/levodopa
	Madopar	Bensarizide/levodopa
Type B MAO inhibitors	Deprenyl	Selegiline
	Eldepryl	Selegiline
COMT inhibitors	Comtan	Entacapone
	Tasmar	Tolcapone

Figure 2. Commonly prescribed anti-Parkinsonian drugs (Hermanowicz, 2001; Yorkston, Miller, & Strand 1995).

Each drug category has a differential role in the treatment of Parkinson's disease. Figure 3 taken from Yorkston et al. (1995) explains the function of each drug category in order to balance the dopamine-acetylcholine relationship.

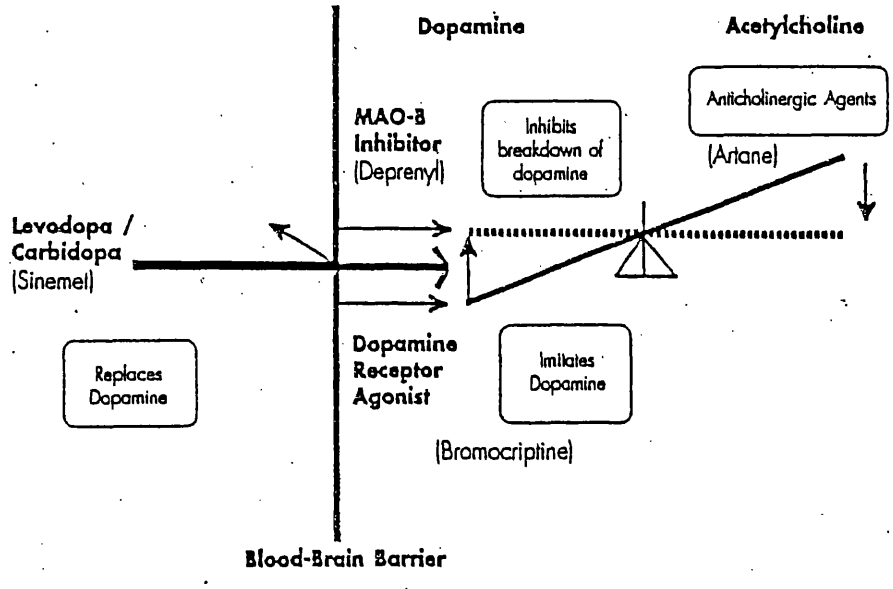


Figure 3. Action of drugs to balance dopamine and acetylcholine systems.

During the history of Parkinson's disease three major categories of drugs were used: anticholinergics, dopamine agonists, and levodopa. The other drug categories (Type B MAO inhibitors and COMT inhibitors) are considered either neuroprotective or complementary to levodopa. The present discussion will give more emphasis on the three major categories of drug treatment while some comments will be spared for the other categories.

Selegiline as a type B MAO inhibitor was successfully used on animals to protect them from induced Parkinsonism (MPTP). The use of selegiline in humans after 1989 in the US aimed to decrease the dopamine breakdown in the brain and increase dopamine in the synapses (Hristova & Koller, 2000). As a result, the early use of selegiline might slow the progression of the disease and prevent nigral neuronal destruction. The results showed that the patients could benefit from selegiline during the first year of treatment (Parkinson Study Group, 1989; Parkinson Study Group, 1993). There is, however, a controversy between researchers if selegiline is protective to the patients. Some researchers favour its use (Olanow, 1992) while others not (Wills, 1998). Recent evidence does not support these findings. Selegiline seems to have a little or no effect in the reduction of Parkinsonian symptomatology (Hermanowicz, 2001; Hristova & Koller, 2000; Oertel & Quinn, 1997). Furthermore, many neurologists in the U.K avoid its prescription because of increased mortality rates in patients who used it in combination with levodopa (Lees, 1995).

COMT inhibitors are used to delay the peripheral breakdown of levodopa by the enzyme COMT and so to increase the duration of levodopa in the brain (Hermanowicz, 2001). They are used as complementary drugs to levodopa to reduce motor complications and to increase the effectiveness of levodopa but

they are not used as early monotherapy (Hristova & Koller, 2000).

Anticholinergic agents (the older symptomatic therapy for Parkinson's disease) and amantadine are used to create a balance of dopamine and acetylcholine. Because of the lack of dopamine in Parkinson's disease the balance of dopamine and acetylcholine is shifted towards acetylcholine. Suppression of the increased acetylcholine through the use of anticholinergics is thought to restore this balance. Nowadays, the use of anticholinergics is still frequent because of their low cost and because of their influence on resting tremor (Hermanowicz, 2001; Koller, 1992; Oertel & Quinn, 1997). However, they do not seem to be so effective for bradykinesia (Koller, 1992). Some authors state that the effectiveness of anticholinergics on tremor is not better than that of levodopa (Hermanowicz, 2001; Hristova & Koller, 2000). A disadvantage of anticholinergics is that they produce serious side effects such as memory problems, lack of concentration, confusion, and hallucinations (Ahlskog, 1996; Koller, 1992; Oertel & Quinn, 1997).

As with other anticholinergics, amantadine blocks the acetylcholine receptors. It is not considered to be the most beneficial drug for the maximum alleviation of Parkinsonian symptomatology (Hermanowicz, 2001). Nowadays, it is used to reduce motor fluctuations such as choreic movements that are produced as a side effect from the prolonged use of levodopa (Hermanowicz, 2001). Wills (1998) states that amantadine and anticholinergics as alternatives to dopamine agonists when used in combination with levodopa can either increase dopaminergic transmission (amantadine and levodopa) or relieve tremor and rigidity (anticholinergics and L-DOPA).

Dopamine agonists work as adjuncts to levodopa or as a synthetic

levodopa. They act directly on post-synaptic dopamine receptors without requiring transformation or facilitation transport across the blood-brain barrier (Hermanowicz, 2001; Hristova & Koller, 2000; Koller, 1992; Koller & Rueda, 1998; Tolosa, Martí, Valldeoriola, & Molinuevo, 1998). Dopamine agonists are used both as monotherapy and as complementary therapy to levodopa-carbidopa to reduce motor fluctuations and dyskinesias (Hermanowicz, 2001; Oertel & Quinn, 1997). Fukuyama, Kawamura, Akigushi, Kimura, and Imai (1996) evaluated bromocriptine (the most extensively used dopamine agonist) monotherapy during the early stage of Parkinson's disease (Hoehn and Yahr stage I or II) and found that 70% of patients benefited from this therapy. They concluded that the use of this therapy might be beneficial in an early stage of the disease. Olanow (1992) suggests the use of dopamine agonists to minimise the levodopa dosage. In contrast, Ahlskog (1996) supports the notion that the early use of dopamine agonists should not be recommended. Because of their high cost, dopamine agonists can be reserved for patients with more advanced disease and motor fluctuations. The disadvantages of their use involve side effects (visual hallucinations and orthostatic hypotension) in elderly patients (Hermanowicz, 2001) and a reduced effectiveness over time when compared to levodopa.

There is general agreement that the most used and most effective agent for the treatment of dopamine deficiency in Parkinson's disease is levodopa. Levodopa is not only widely used, but also its responsiveness to the motor symptomatology is considered as a part of the diagnosis of Parkinson's disease (Koller, 2000). Levodopa is well tolerated by most patients, and its use tends to alleviate symptoms such as bradykinesia and rigidity while it has a variable effect

on tremor (Koller, 1992). It is the hallmark of pharmacological treatment in idiopathic Parkinson's disease and it is considered effective for both long and short-term duration. Its short duration effect is more prominent with chronic therapy but some patients who are starting the therapy show an immediate improvement. The mortality rate of Parkinsonian patients has been reduced due to levodopa medication (Agid et al., 1999).

The premature decarboxylation (breakdown) of levodopa to dopamine by gastric enzymes might create side effects. These involve primarily nausea and postural hypotension and after a prolonged use, motor fluctuations ("on-off") and dyskinesias. The initiation of carbidopa in early Parkinson's disease hoped to counter nausea, to potentiate the effect of levodopa and to decrease the percentage of levodopa that needs to be orally administered by 75% (Ahlskog, 1996; Nutt, Hummerstad, & Gancher, 1992; Oertel & Quinn, 1997). A combination of levodopa-carbidopa (Sinemet) has been used in North America while a combination of levodopa-benserazide (Madopar) has been used outside North America. Carbidopa and benserazide inhibit the dopa decarboxylase enzyme and so increase the effectiveness of levodopa (Hristova & Koller, 2000). Adding sodium, controlled-released levodopa (CR), and dopamine agonists may also help to improve postural hypotension, motor fluctuations, and dyskinesias, respectively (Koller, 2000).

Controlled-released combinations of levodopa continuously stimulate dopamine receptors and so lessen motor complications (Hristova & Koller, 2000). The advantages of controlled-released levodopa-carbidopa depend on the circumstances in which the drug is taken, but include the better absorption by an empty stomach and the minimization of nausea if the drug is given with meals

(Pahwa & Koller, 1996). The doses should not be more than 300-400 mg/day split in three equal doses in the early stages of the disease.

Problems arise when clinicians aim to find the best treatment approach for Parkinsonian patients. It is still controversial among researchers if levodopa should be started early or late during the course of the disease. Even though levodopa is characterised as the gold standard for the medication of Parkinson's disease, side effects after some years of its use make researchers seek for alternative treatments in the early Parkinson's disease. Olanow (1992) suggests that levodopa should be an adjunct to selegiline and dopamine agonists when the clinical response is no longer satisfactory in the early treatment for Parkinson's disease. However, there is no evidence that the delay of levodopa will also delay its adverse effects, namely motor fluctuations and dyskinesias (Koller, 1992). The appearance of motor fluctuations is the result not only of the postsynaptic changes that are caused by the long-term intermittent administration of levodopa but also from the lack of dopaminergic neurons in the brain that is increased with time (Agid et al., 1999). Questions have arisen about long-term levodopa use, and whether or not it could cause or accelerate cell death. It is now assumed that levodopa does not create or promote cell death in healthy people or Parkinsonian patients (Agid et al., 1999; Koller, 2000). Because further research is needed to prove that levodopa is not toxic to dopamine neurons over time, it is advisable to be used only by older patients (> 70 years), or, by patients less than 70 years old when symptoms are not controlled by other agents (Agid et al., 1999).

In conclusion, it is at the discretion of the clinical neurologist to decide if levodopa should be started early in the course of the disease, by weighing factors such as the clinical symptoms of the patient, patient's needs, and the

neurologist's personal experience. Other opinions favour the combination of levodopa-carbidopa as the best choice for initial treatment based on patients needs (Ahlskog, 1996; Hristova & Koller, 2000; Scientific American Magazine, 1997; Wills, 1998). The use of drugs other than levodopa in the early treatment of Parkinson's disease, such as selegiline (to help patients to stabilize the condition of the disease) and dopamine agonists (to help patients control motor fluctuations), has a less clear role during the first year of the disease (Ahlskog, 1996). The opposite opinions follow a more "conservative" way by using dopamine agonists in the beginning of therapy and reserving levodopa until the disease progresses (Agid et al., 1999; Oertel & Quinn, 1997).

1.4.1 The effect of medication on dysarthria of Parkinson's disease

Few studies have investigated the effect of medication on hypokinetic dysarthria of Parkinson's disease. They are characterised by heterogeneity in terms of both the neurological stages of Parkinson's disease in their subjects and their instrumentation. It is probably this heterogeneity that has given rise to conflicting results in these studies. Among them, the older studies showed a tendency for improvement of speech after levodopa treatment while the newer studies did not confirm these findings (Schulz & Grant, 2000).

Historically, the advent of the levodopa era in the late sixties led to studies that searched the effect of this new medication on speech and voice (perceptually and instrumentally). In the nineties, most of the studies used kinematics and seemed to give more emphasis on the force of the articulators during speech and non speech tasks. In addition, speech and voice were investigated under new drug combinations. Because the number of all studies

that examined speech and voice before and after medication is rather small and because these studies involve different instrumentation and different drugs, a detailed description of them will follow.

Two fairly recent studies were found to investigate the effect of type B-MAO inhibitors (selegiline) on speech and voice. One study measured 40 variables in respiration, phonation, resonance, articulation, and prosody in 10 patients with a moderate disease (expressed by the stage 3 in Hoehn and Yahr scale) and a duration of disease of 6-12 years (Shea, Drummond, Metzer, & Krueger, 1993). The tasks involved diadochokinesis, sustained phonation, and reading. The variables that showed significant improved differences before and after selegiline treatment involved respiration (vital capacity), rate, and diadochokinesis (labial pursing during 5 consecutive 'oo-ee', 5 lingual protrusions and retractions, 5 lingual elevations and depressions, and 5 diadochokinetic productions of 'ka-ta'). The other study perceptually examined the speech and voice abnormalities of 12 non depressed and non demented patients with early Parkinson's disease (the duration of disease ranged from 18 months to 5 years and 10 months) before medication and after medication with Deprenyl (Stewart, et al., 1995). No improvement was found after medication. The results showed that all subjects exhibited at least two perceptual characteristics of mild dysarthria before and after medication. The inconsistencies in the results of these studies are probably due to the heterogeneity (different neurological stage and duration of disease) between their samples.

There are no studies to date, which have investigated the effect of anticholinergics on speech and voice of Parkinson's disease. One report in the

early eighties referred that anticholinergics do not seem to improve speech (Critchley, 1981). This is probably logical because their use aims to relieve limb tremor (Hermanowicz, 2001; Koller, 1992; Oertel & Quinn, 1997). If there were to be any effect of anticholinergics this would probably be on voice tremor.

Most of the studies that investigated the effect of medication on hypokinetic dysarthria used levodopa as a primary treatment in their subjects. Some of them took place in the early seventies and focused on the effect of dopatherapy on labial musculature, articulation timing, intelligibility, rate, and voice quality. The results of these studies showed an improvement after levodopa medication on lips, clarity of speech, articulation timing, voice quality, and pitch (Leanderson, Meyerson, & Persson, 1971; Mawdsley & Gamsu, 1971; Nakano, Zubick, & Tyler, 1973; Wolfe, Carvin, Bacon, & Waldrop, 1975). With the exception of two studies (Cahill et al., 1998; Gallena, Smith, Zeffiro, & Ludlow, 2001), newer studies do not support these results (Daniels, Oates, Phyland, Feiglin, & Hughes, 1996; Gentil, Tournier, Perrin, & Pollak, 1998; Gentil, Tournier, Pollak, & Benabid, 1999; Poluha, Teulings, & Brookshire, 1998). Metter and Hanson (1986) suggest a variable effect of levodopa on speech in one patient with Parkinson's disease. When "off" levodopa the patient showed a decrease in articulatory precision (shown in a worse intelligibility score), prosody, relative intensity, and a slower rate. In contrast, mean fundamental frequency and dysphonia did not show changes.

The studies that employed levodopa on labial movement gave conflicting results. Two earlier, and one recent study showed an improvement in labial movement (Cahill et al., 1998; Leanderson et al., 1971; Nakano et al., 1973) while two recent studies showed no difference after levodopa medication (Gentil

et al., 1998; Gentil et al., 1999).

Leanderson et al. (1971) examined the effect of levodopa on labial musculature before and after medication using electromyography (EMG). Seven Parkinsonian patients with dysarthria produced VCV utterances. The results showed an improvement of speech in six patients when their recorded speech samples were compared before and after medication. The after medication results were accompanied by live facial expression, smooth and fast lip movements, the re-establishment of reciprocal muscular activation, and a reduction in muscle background activity.

Nakano et al. (1973) examined speech intelligibility and labial movement in 18 patients and in four conditions: no treatment, placebo, procyclidine hydrochloride, and levodopa. An intelligibility subtest was given before and after treatment and speech samples were recorded. The results showed an improvement in intelligibility and labial movement (11 out of 18 subjects) after the administration of levodopa and no significant improvement after the administration of procyclidine hydrochloride compared to placebo. The improvement of labial movement was manifested as a decreased latency between the initiation of labial movement and speech, with increased speed and symmetry.

Cahill et al. (1998) examined the effect of levodopa therapy on lip strength and endurance in 16 subjects with variable duration of disease, neurological stage, and age across the levodopa cycle. Lip pressures in non speech tasks involved maximum pressure and maximum sustained lip pressure (squeezing the lips for 7 seconds), fine lip pressure (standard deviation of pressure changes when the subject compressed the lips for 5 seconds at 50% of maximum lip

pressure), repetitions of maximum lip pressure with a rate of one per second (mean pressure over 10 repetitions), and maximum rate of repetitive lip pressure (number of repetitions in 10 seconds). Lip pressures in speech tasks (2 sentences measuring pressures of [p]) were recorded. All pressures were measured with a use of a pressure transducer. The results showed an increased performance of lip measures (maximum and maximum sustained lip pressure and fine lip pressure at 50% of maximum pressure) in non speech and speech tasks after 1.5 hour of medication.

Two studies measured the effect of dopatherapy alone (Gentil et al., 1998) and dopatherapy as compared to subthalamic nucleus stimulation (Gentil et al., 1999) on intelligibility (Unified Parkinson Disease Rating Scale - UPDRRS), force finger, and force movements of the articulators (upper and lower lips and tongue). These studies used a pressure transducer during non speech tasks to record changes. The results of both studies showed no effect of dopatherapy on force of the articulators, while a significant result was found on force finger. The authors emphasised that the differences of the effect of medication on forefinger force as compared to no differences in the force of articulatory organs, might reflect that the orofacial system involve other mechanisms resulting from non dopaminergic lesions. However, further research is needed to verify this hypothesis for many reasons. First, the above studies employed subjects with moderate disease involvement and motor fluctuations. It is possible that after many years of treatment the orofacial system does not respond well to levodopa (as occurs in all mobility symptoms of Parkinson's disease). Second, the scoring scale of intelligibility was not so sensitive to extract accurate measurement. Third, it is unknown if lip and tongue pressures

as measured by a pressure transducer correlate with dysarthria characteristics in running speech.

One study (Wolfe et al., 1975) perceptually evaluated the speech and voice of 17 patients with different forms of Parkinsonism (7 patients with idiopathic Parkinson's disease, 7 patients with postencephalitic Parkinsonism, and 3 patients with arteriosclerotic Parkinsonism). Articulation, voice quality, pitch variation (inflectional changes), and rate before and after levodopa administration were measured. Significant improvements after medication were found in voice quality, pitch variation, and articulation while no significant differences were found in the rate of speech, even though there was a tendency for the patients to speak faster after treatment. Voice quality was the most affected factor of mild to moderate dysarthria pre and post treatment with levodopa. A significant positive correlation of improvement in speech was also found when compared to the amount of improvement in physical findings. In the same study, another parameter that influenced the response to levodopa was the duration of the disease. Two patients that exhibited the greatest amount of improvement of speech had the disease for only one year. Both short-term and long-term levodopa treatment had a positive influence on speech and voice even though the number of patients who were examined on a long-term basis was too small (only 4 subjects), to permit generalisations.

Two studies emphasised the duration of phonation and the vocal characteristics of Parkinsonian patients after the administration of levodopa (Daniels et al., 1996; Mawdsley & Gamsu, 1971). Mawdsley and Gamsu (1971) examined the duration of phonation and the duration of pauses in 20 Parkinsonian patients (16 patients with Parkinson's disease) while the patients

were counting from 1-10 in two conditions: before medication and after medication. The results showed no improvement in the rate of speech, but the production of each digit during counting took a shorter time after medication, and it was separated from the previous and next productions by lengthened pauses. The authors emphasised that this improvement occurred only in the patients with idiopathic Parkinson's disease and not in the other four postencephalitic Parkinsonian patients.

Daniels et al. (1996) did not find any perceptual or acoustic differences in intensity, variability of fundamental frequency and intensity, whisperiness and harshness in 30 subjects with idiopathic Parkinson's disease while sustaining an [a] and while conversing for 30 seconds. All subjects exhibited dysphonia and motor fluctuations. Even though the authors conclude that vocal dysfunction is probably not related to nigrostriatal dopamine deficiency their results may be caused by the non response of dopatherapy to voice symptomatology due to advanced state of the disease (existence of motor fluctuations).

Finally, one study used electromyography (EMG) to examine voice onset and offset after levodopa therapy in Parkinsonian patients not previously medicated (Gallena et al., 2001). The results showed a beneficial effect of levodopa after therapy. Before medication, laryngeal bowing of the vocal folds was associated with increased activity of thyroarytenoid muscle (TA) and cricothyroid muscle (CT) to a lesser degree, but the reverse was found after levodopa treatment. The activity of thyroarytenoid decreased and breathiness and speech improved (measured perceptually). In normal voicing the combined contraction of TA and CT is to increase pitch while TA shortens vocal folds and closes the vestibule of larynx, and CT stretches vocal folds. The results of this

study confirm that vocal fold bowing is a manifestation of rigidity. However, the amount of levodopa that was given to the untreated patients was too high (250-300 mg) for patients who were not previously treated with levodopa. It is possible that this higher amount of levodopa decreased the laryngeal muscles' activity. In early Parkinson's disease the insertion of levodopa follows a consistent way starting with smaller doses (100 mg). It is not known what the results on muscle activity would be if smaller doses would be given. However, this assumption does not preclude the fact that levodopa produced a beneficial effect on voicing in this study.

Problems in speech and voice after chronic use of levodopa have been reported. Marsden and Parkes (1976) reported "on-off" effects in 15-40% of patients with Parkinson's disease treated with levodopa medication for 2-3 years. These effects were defined as fluctuations of activity and they occurred sometimes more than 3 times per day. Clinical observations of "on-off" effects showed early-morning akinesia, freezing episodes (i.e., a sudden immobility during a difficult task), end-of-dose deterioration, peak dose dyskinesia (hyperkinetic involuntary movement as in tic or spasm) which was accompanied by oromandibular dystonia (sustained muscle contractions causing twisting and repetitive movement or abnormal postures) often causing difficulties in speaking or swallowing and peak dose akinesia. In contrast, Critchley (1976) reports the appearance of dysphonias or aphonias with simultaneous peak-dose akinesia. The akinesia and dysphonia, although rare, were associated with the disappearance of rigidity and tremor at the peak of levodopa action. Duffy (1995) states that the side effects of anti-Parkinsonian medication involve dystonia, dyskinesias, confusion, and symptom fluctuations during a dosage cycle.

According to him, the "on-off" effects may determine the appearance of hyperkinetic dysarthria or the deterioration of hypokinetic dysarthria.

The so-called "Yo-Yo-ing" effect happens when the above effects occur many times during the day. During this state, the voice becomes soft, the speech becomes unintelligible, walking and standing become impossible. The symptoms may occur for a few seconds or minutes and thereafter disappear. "Yo-Yo-ing" is characterised by unpredictability, occurs after 1-3 years of levodopa treatment and seems unrelated to the time of dosage. Also, it occurs in patients who have achieved a good response to treatment and it is not treated successfully. "Yo-Yo-ing" may be due to excessive levodopa dosage and/or progression of the disease (Marsden & Parkes, 1976). The influence of levodopa on speech and voice denotes the key role of neurotransmitter substances (it is already known that levodopa replaces the neurotransmitter dopamine) to speech and voice (Critchley, 1981).

Most of the studies examining the effects of levodopa treatment on dysarthria are characterised not only by different use of instrumentation but also by a variable duration of disease and neurological stage among their patients. This may be a confounding factor for the investigation of the effect of dopatherapy on speech and voice because the motor fluctuations may determine the effect to therapy. The existence of motor fluctuations is an unexplained phenomenon. They may be caused by both chronic levodopa use and dopaminergic cell loss due to the progression of the disease. In order to draw conclusions about the effect of medication on speech and voice, the existence of homogeneous populations in neurological stage and duration of disease is needed. The present study aims to accomplish this purpose by investigating

dysarthria in subjects only recently diagnosed with idiopathic Parkinson's disease (Hoehn and Yahr stage I).

1.5 Perceptual and acoustic studies of hypokinetic dysarthria

In the past, the identification of different types of dysarthria was based primarily on perception. Today, perceptual methods are still the primary tool in the assessment of motor speech disorders, but are followed by acoustic methods. However, inconsistencies have arisen after attempts to classify dysarthria using different methods (perceptual, neurological, or site of lesion). Netsell (1986) supports the notion that case studies with highly restricted, well-documented lesions to pathways or modules of the speech motor system may be more effective in connecting different types of dysarthria with an associated lesion. In the present study, a review of the literature in hypokinetic dysarthria that was diagnosed with either perceptual or acoustic methods, or both, will be given.

1.5.1 Prominent perceptual studies of hypokinetic dysarthria

Before a description of prominent perceptual studies in Parkinson's disease will take place, it is noteworthy to mention the work by Laver (1980). He was the first to attempt to emphasise the importance of phonetic description of voice quality (Green & Mathieson, 1997; Wirz & Beck, 1995). For Laver (1980) the study of phonetic components of voice quality is related to the study of spoken language. In contrast to other researchers, he conceived voice quality in a broad sense to mean the characteristic auditory colouring of an individual speaker's voice. He classified phonation types (harsh, whispery, breathy, creaky,

falsetto and modal) and gave tension ratings in the positions of labial, mandibular, lingual, velopharyngeal and laryngeal structures. The so-called Vocal Profile Analysis (VPAS) (Laver, 1991) was important to show the variety of vocal qualities and phonetic gestures possible, and it is a useful indicator of vocal features of dysphonia (Greene & Mathieson, 1997).

Laver's work is more holistic than other methods of perceptual assessment of voice (Buffalo Profile of Voice Disorder, Missouri Profile of Voice Disorder, and GRBAS). It includes factors such as replicability, interjudge reliability and unambiguous definitions of to what constitutes voice (Wirz & Beck, 1995). The contribution of the whole vocal apparatus to voice quality, the comparison of all vocal features with a specified neutral baseline and the overall impression of voice quality as derived by various potentially independent components make his work different from other perceptual schemes of voice assessment (Wirz & Beck, 1995). According to Kent (1997), Laver's approach may be suitable for the description of quality impairments in motor speech disorders.

In neurological disorders, perceptual studies were the first to describe dysarthria and voice symptomatology. The perceptual studies by Darley and his colleagues in the late sixties and seventies (Darley et al., 1969a, 1969b, 1975). The perceptual studies by Darley and his colleagues in the late sixties and seventies (Darley et al., 1969a, 1969b, 1975) are used as a reference in any current study of dysarthria. As a result of their work, distinctive patterns of dysarthria were established and associated with each neurological disorder. Darley et al. (1969a) perceptually rated patients (N = 212) on seven neurological disorders in a number of dimensions of speech and voice, using a 7 point scale

of severity (from 1 = normal to 7 = very severe). Figure 4 below shows the speech characteristics and their dimensions on which patients were rated.

SPEECH CHARACTERISTICS	DIMENSIONS
Pitch	Pitch level, pitch breaks, monopitch, voice tremor.
Loudness	Monoloudness, excess loudness variation, loudness decay, alternating loudness, loudness level (overall).
Vocal quality	Harsh voice, hoarse (wet) voice, breathy voice (continuous), breathy voice (transient), strained-strangled voice, voice stoppages, hypernasality, hyponasality, nasal emission.
Respiration	Forced inspiration-expiration, audible inspiration, grunt at end of expiration.
Prosody	Rate, phrases short, increase of rate in segments, increase of rate overall, reduced stress, variable rate, intervals prolonged, inappropriate silences, short rushes of speech, excess and equal stress.
Articulation	Imprecise consonants, phonemes prolonged, phonemes repeated, irregular articulatory breakdown, vowels distorted
General Impressions	Intelligibility, bizareness

Figure 4. Perceptual speech characteristics and speech dimensions (Darley et al., 1969a).

Detailed definitions of the most important dimensions are given in Appendix A. Appendix A also includes medical and other terms that were judged necessary to be defined. A review of prominent perceptual studies of hypokinetic dysarthria follows.

In Darley et al. (1969a), the speech characteristics that were found to be more deviant in Parkinsonism were, in order of severity (from most severe to

least severe): monopitch, reduced stress, monoloudness, imprecise consonants, inappropriate silences, short rushes, harsh voice, breathy voice (continuous), low pitch, and variable rate. The combination of monopitch, reduced stress, and monoloudness according to the authors presented the most striking phenomenon in Parkinsonism. The researchers also reported that the alternating speech movements in the Parkinsonian group were reduced in range and tended to become progressively smaller even though at times they were perceived as slow. The imprecise consonants were considered to be the result of reduced excursion of the articulators, rather than the variability of the rate. Moreover, imprecise consonants showed the highest correlation with intelligibility (0.91) followed by short rushes of speech (0.79) and reduced stress (0.78) while monoloudness showed an even lower correlation (0.60) with intelligibility.

Darley et al. (1969b) correlated the 10 prominent deviant dimensions of each neurological group in order to find possible "clusters". The results for Parkinsonism showed one distinctive cluster. The authors named this cluster "prosodic insufficiency" and they hypothesised that it was again due to the reduced range of movements, a characteristic of Parkinsonism. This cluster involved:

- Monopitch
- Monoloudness
- Reduced stress

and extended to include

- Short rushes of speech
- Variable rate
- Imprecise consonants.

The authors reported that the extent of this cluster was unique to Parkinsonism when compared to the other neurological groups and it reflected its neuromuscular quality (the fast repetitive movements of restricted range). In addition, while single movements as in a sustained [a] tend to be slow, the repetitive movements as in [pa] produced at a fast rate tend to be either slow or abnormally fast, with a very limited range. Decay of loudness or decreased loudness were dimensions that were found only in Parkinsonism compared to the other neurological disorders and were not correlated with the above-mentioned cluster. In general, Darley et al. (1975) emphasise that respiratory, phonatory, and articulatory muscles are limited in excursion and produce prosodic dysfunction, the most prominent feature of hypokinetic dysarthria.

Recently, Duffy (1995) reviewed the perceptually distinguishing speech, speech-related findings, and oral-mechanism findings in hypokinetic dysarthria. Duffy added dimensions, such as physical characteristics and patients' complaints to the overall description of hypokinetic dysarthria. Figure 5 below, shows his work.

Phonatory-Respiratory	Reduced loudness.
Articulatory	Repeated phonemes, palilalia, rapid or "blurred" alternating motion rates (AMRs).
Prosodic	Reduced stress, monopitch, monoloudness, inappropriate silences, short rushes of speech, variable rate, increased rate in segments, increased overall rate.
Physical	Masked facial expression, tremulous jaw, lip, tongue, reduced rate of motion in AMR tasks, head tremor.
Patient complaints	Reduced loudness, rapid rate, "mumbling", "stuttering", difficulty initiating speech, stiff lips.

Figure 5. Primary speech and speech-related findings in hypokinetic dysarthria (Duffy, 1995).

Similar results to the results of the studies by Darley et al. (1969a, 1969b) have been reported (Chenery, Murdoch, & Ingram, 1988; Logemann et al., 1978). Aronson (1990) reports that reduced loudness and breathy voice quality might be early signs of hypokinetic dysarthria. In contrast, Critchley (1981) supports the notion that the initial defect in the untreated Parkinsonian patient involves an inability to control respiration for speech that progresses to include phonation and articulation. Chenery et al. reported the prominence of abnormalities in phonation followed by respiration and articulation in the context of reading a passage. Imprecise articulation accounted for approximately 85% of the variance in overall intelligibility while loudness variation contributed 10%. The results of the Frenchay Dysarthria Assessment (FDA) in the study by Chenery et al. showed similar patterns. Larynx, tongue mobility, and mild disturbances in the movement of lips and jaw were found in a descending order of severity (most severe to less severe). However, no information about the duration of the

disease was reported in this study.

Because of the size of its sample, the study by Logemann et al. (1978) occupies a special position in the research of dysarthria in Parkinsonism. The authors examined the frequency and the patterns of vocal tract dysfunction in 200 Parkinsonian patients (idiopathic or postencephalitic) in all five stages of the disease. All patients were withdrawn from medication for 2 weeks or they were newly diagnosed. The patients read the sentence version of the Fisher-Logemann test of articulation competence and conversed for 3-5 minutes. Speech samples were recorded and were listened to by 2 trained listeners for rate, voice, resonance, and articulation disorders. Laryngeal disorders (breathiness, roughness, hoarseness, and tremulousness) were found in 89% of the patients, followed by articulation disorders (lingual, labial, or both) that were found in 45% of the patients. Disturbed rate and disturbances of resonance (hypernasality) were found in 20% and 10% of the patients, respectively. According to the authors, the typical Parkinsonian patient is highly like to have impaired voice quality and an articulation disorder. Further analysis of the co-occurrence of vocal tract dysfunction showed that 45% of the patients showed laryngeal dysfunction without articulatory errors while the remaining showed laryngeal dysfunction together with articulatory errors. In this study, no analysis took place of the speech/voice abnormalities and neurological stage of Parkinson's disease.

1.5.2 Studies examining respiration

Physiological measurements of respiratory parameters that did not involve speech showed that the respiratory dysfunction in Parkinson's disease (PD)

becomes evident after neurological stage 3 in Hoehn and Yahr scale (De Pandis et al., 2002; Hovestadt, Bogaard, Meerwaldt, Meché, & Stigt, 1989). In fact, the major inclusion criterion in these studies was the existence of at least stage 3 in Hoehn and Yahr scale. Respiratory dysfunction is considered asymptomatic or at least infrequent in early Parkinson's disease (Shill & Stacy, 1998).

Studies that measure respiration on speech tasks showed the same trend including Parkinsonian subjects with either a disease duration of more than 5 years (Lethlean, Chenery, & Murdoch, 1990) or at least stage 3 in Hoehn and Yahr scale (Murdoch, Chenery, Bowler, & Ingram, 1989; Solomon & Hixon, 1993).

Murdoch et al. (1989) report task specific respiratory abnormalities. Irregularities that were found in chest wall movements during vowel prolongation and syllable repetition were not shown in reading and conversation. Also, a greater breathing rate and minute ventilation was found in Parkinsonian subjects as compared to the controls as well as small rib cage volumes and abnormally large abdominal volumes at the initiation of speech (Solomon & Hixon, 1993). The rigidity of the respiratory muscles was suggested as the primary cause of respiratory abnormalities (Duffy, 1995; Murdoch et al., 1989) and was followed by hypokinesia and difficulty initiating speech movements (Duffy, 1995).

The findings of the above studies may be confounded by the involvement of subjects in advanced neurological stage of the disease and by the effect of motor complications that take place at this stage. Individual variation among subjects in respiratory features (Theodoros & Murdoch, 1998) may also be a confounding factor in the explanation of the results. Accordingly, Duffy (1995) reports that the contribution of certain breathing abnormalities (reduced vital

capacity, reduced amplitude of chest wall movements, irregularities in breathing patterns and increased respiratory rates) to speech of Parkinson's disease is not clear.

In conclusion, it appears that respiration for speech/voice is not affected at the beginning of Parkinson's disease (PD). The existence of phonatory problems in early PD is probably not due to respiratory abnormalities. This is supported by findings where tracheal pressure was found normal between PD and control subjects while the oral pressure in the PD subjects was smaller (Solomon & Hixon, 1993). Also, this fact is supported by the absence of studies that measure the effect of respiration on speech in early PD and by the existence of studies that show respiratory problems in advanced neurological stages of the disease (De Pandis et al., 2002; Hovestadt et al., 1989).

1.5.3 Studies examining phonation

It is without doubt that the phonatory system in hypokinetic dysarthria is being affected by the process of the disease. Perceptual studies have investigated vocal quality, pitch, and loudness, while acoustic studies have investigated the correlates of maximum sustained phonation time, fundamental frequency and intensity. More specifically, in acoustic studies, average fundamental frequency, standard deviation of fundamental frequency, jitter and shimmer have been employed to correlate with vocal quality. Different aspects of fundamental frequency and intensity have also been used during reading and monologue/conversation. Because the present study uses a different type of instrumentation (electrolaryngography) and a limited number of electrolaryngographic studies in hypokinetic dysarthria exist, an overview of

acoustic studies that focused on all of the above measures will be presented here. A review of electrolaryngographic studies on hypokinetic dysarthria will follow in the next section. Finally, other physiological studies on hypokinetic dysarthria will be used in the literature review but their reference in the current study will not be exhaustive.

Although Darley et al. (1975) did not report hoarseness to be a deviant speech dimension, other perceptual studies emphasise its existence in a range of 33-100% of their sample (Chenery et al., 1988; Logemann et al., 1978; Ludlow & Bassich, 1984). Harshness has been found in 77-84% of their sample (Chenery et al., 1988; Ludlow & Bassich, 1984; Zwirner & Barnes, 1992). Some studies have found breathiness to occur in 50-95% of hypokinetic dysarthric speakers (Chenery et al., 1988; Zwirner & Barnes, 1992) or being double compared to normal controls (Ludlow & Bassich, 1983), while others have found it to a lesser degree (Logemann et al., 1978; Ludlow & Bassich, 1984). Levodopa medication may play a role in the variable incidence rates of breathiness among subjects (the subjects' voices are becoming closer to hyperkinetic) but this is a hypothesis that needs to be tested (Adams, 1997; Ludlow & Bassich, 1984). Glottal fry and vocal tremor (as a manifestation of deficits in pitch steadiness) have been perceived in 68% of cases (Chenery et al., 1988). However, Holmes, Oates, Phyland, and Hughes (2000) perceived tremor as a feature of late disease rather than early Parkinson's disease.

Acoustic evidence seems to support the perception of impairment of vocal quality in hypokinetic dysarthria. Vocal quality has been investigated through sustained phonation that is frequently used in order to minimise the effects of articulatory adjustments and to isolate the phonatory system (Zwirner, Murry, &

Woodson, 1991). Reduced duration in sustained phonation time has been found in Parkinsonian patients due to phonatory inefficiency and disturbed vocal quality (Canter, 1965a; Ludlow & Bassich, 1983). Increased average fundamental frequency has been found in some studies (Gamboa et al., 1997; Hertrich & Ackermann, 1995; Ludlow & Bassich, 1983; Ludlow, Coulter, & Gentges, 1983; Ramig, Scherer, Titze, & Ringel, 1988) while no significant differences were found in one study (Kent et al., 1994). Two of these studies (Hertrich & Ackermann, 1995; Gamboa et al., 1997) found the increased mean fundamental frequency only in the male subgroup of Parkinsonian patients compared to controls. The different findings in the study by Kent et al. may be due to the fact that no matching in age and duration of disease occurred in his Parkinsonian subjects compared to control subjects.

Standard deviation of fundamental frequency (SDFo) is used to evaluate phonatory stability in hypokinetic dysarthria (Zwirner & Barnes, 1992). SDFo has been proposed in studies that investigated normal voicing and ageing as a better predictor of vocal age (Linville, 2000). It has been used as a better approach to fundamental frequency range to measure the average distance of values from the mean as an index of variability (Baken, 1997). SDFo as a measure of a long term phonatory instability (differentiated from jitter and shimmer as measures of short term phonatory instability) has been found to increase (Zwirner & Barnes, 1992; Zwirner et al., 1991). However, no statistical significance was found in these studies.

Perceptually, the pitch in hypokinetic dysarthria has been reported to be monotonous and the pitch range (variability) restricted with more limited pitch inflections (Darley et al., 1969a, 1969b, 1975; Gentil & Pollak, 1995). Acoustic

evidence supports the perceptual impressions. In reading and monologue, increased mean fundamental frequency has been found in the Parkinsonian patients (Canter, 1963; Holmes et al., 2000; Ludlow & Bassich, 1983, 1984). Holmes et al. report that increased mean fundamental frequency is attributed to the late stage of the disease while no differences were found in patients with early Parkinson's disease compared to controls.

Duffy (1995) discusses the discrepancy on pitch between perceptual and acoustic studies. The perceived pitch in the study by Darley et al. (1969a) was reported to be low while the aforementioned acoustic studies found a higher fundamental frequency. According to Duffy, this discrepancy may reflect intersubject variability among these studies or the insensitivity of mean fundamental frequency itself (not in combination with other measures) to distinguish features of hypokinetic dysarthria. Other factors may also contribute to these discrepancies.

One such factor is the issue of gender differences. Gender differences in relation to perceived pitch have also been reported to normal voice production (Baken, 1997; Beck, 1997; Green & Mathieson, 1997; Linville, 2000). In the ages 60-69 (where between the mean age of appearance of Parkinson's disease occur) the average speaking fundamental frequency in normal voice production during reading in males is 112 Hz (Baken, 1997; Greene & Mathieson, 1997) while in females is 199 Hz (see a summary of relevant studies in Baken, 1997).

Beck (1997) supports the notion that average fundamental frequency (Fo) lowers during the life span (from childhood to adulthood) but in a different way for the two sexes. Different patterns of physiological changes seem to be the cause of these sex differences. Furthermore, considerable individual variation

exists.

In females, there is a slight drop in fundamental frequency in older people. A possible cause of this drop may be a generalised loss of muscle tone, ossification of laryngeal cartilages and hormonal changes. Hormonal changes are the possible reason that women may show a rise in fundamental frequency around the age of 50 years and a drop thereafter (Green & Mathieson, 1997; Linville, 2000).

In contrast, fundamental frequency and age in males do not have a straightforward relationship. A slight increase in fundamental frequency after the sixth decade of life has been reported, reaching the highest level by the age of 85+ (Linville, 2000), and is due to possible stiffness of the vocal folds (Beck, 1997; Linville, 2000). Anatomical changes in an increased rate than in females occur in older males due to ossification and calcification of cartilages, erosion of joint surfaces and thinning of the lamina propria (Linville, 2000).

Three studies showed gender specific differences where the male Parkinsonian subjects exhibited increased mean fundamental frequency while the female subjects did not compared to controls (Gamboa et al., 1997; Hertrich & Ackermann, 1995; Kent et al., 1994). However, these results do not seem to be consistent (no statistical significance was reached in Kent et al. study). Hertrich and Ackermann (1995) discuss that the gender differences in their study may be the result of age in the male subjects (mean fundamental frequency was correlated with age). Along the same lines, Holmes et al. (2000) report that the mean speaking fundamental frequency was associated with advanced age in the male group of Parkinsonian subjects with early Parkinson's disease compared to controls.

A restricted variability of fundamental frequency (Fo range) has also been found (Canter, 1963; Gamboa et al., 1997; Ludlow & Bassich, 1983, 1984). In range, Canter (1965a) reported the same results in Parkinsonian subjects compared to controls while they were singing the word [no] at lowest and highest pitches. Using the same technique as Canter, fundamental frequency range (Fo range) as difference in Hz, of an ascending [a] production (between low and high points divided by the mean fundamental frequency) was found to be restricted in the Parkinsonian patients (Ludlow & Bassich, 1983, 1984). The standard deviation of fundamental frequency (SDFo) has been found to decrease in a male Parkinsonian subgroup (Gamboa et al., 1997) and in a female subgroup with late Parkinson's disease (Holmes et al., 2000). Metter and Hanson (1986) found an inverse relation of fundamental frequency variability (expressed as a ratio of the mean standard deviation of fundamental frequency divided by the mean fundamental frequency) to dysarthria severity and clinical disability. In other words, as dysarthria severity increased the fundamental frequency variability decreased. Ludlow and Bassich (1984) found a relationship between monopitch (perceptual measure) and reduced Fo range as measured acoustically on the pitch glide task (low to high pitch glide on [a]). They suggested that the pitch glide task is a valid assessment for monopitch.

In the measurement of "pitch range" in a voice many factors need to be taken into consideration that may limit the application of the results in a study. These include age, gender, race, muscle misuse, personality differences and psychosocial or linguistic code of the person. Some or all of these factors may interact to confound the diagnosis of a voice disorder (Baken, 1997; Rammage, Morrison, & Nichol, 2001). Linville (2000) suggests that the restricted maximum

phonational range in normal voice production is attributed in older women only while in men not. However, she admits that this is a contradictory issue among researchers. Factors such as personality differences and psychosocial differences among the Parkinsonian subjects and their pair matched controls may be limiting factors in the interpretation of the results. For example, psychosocial conflicts and not the neurological disorder itself (Parkinson's disease) may determine a narrow pitch range. Finally, speaking fundamental frequency range is based statistically on extremes and a single instance of a high frequency can determine the upper limit of the range of observed fundamental frequencies even though this might be a momentary "slip of the larynx" (Baken, 1997). The non existence of norms in the Greek language in fundamental frequency range is a limiting factor to the interpretation of the results in the present study. More research is needed to clarify these limiting factors. However, the present study is a first attempt to describe dysarthria and more specifically phonation in a Greek sample at the beginning of Parkinson's disease.

Paralinguistic, sociolinguistic, and extralinguistic factors may also play a role in the measurement of voice (Beck, 1997). Paralinguistic aspects of voice source variation signal a speaker's mood, emotion and attitude to the listener. The sociolinguistic function of voice source variation differentiates voice quality among linguistic, regional and social groups. Finally, extralinguistic factors such as size and shape of the laryngeal structures and physical and mental health may show variation in the voice source that may be reflected in changes in fundamental frequency, intensity and quotients. Although some care was taken to control some of these factors (appearance of dementia and matching of

groups in age, gender, and education), no systematic manipulation of the other factors has been taken.

Baken (1997) supports the use of jitter or frequency perturbation (a measure of variability of fundamental frequency or how a period differs from the period that immediately follows it) to show variability of the fundamental period in the evaluation of laryngeal and vocal pathologies. Although jitter cannot be used as a sole diagnostic criterion, it is a sensitive measure for pathological changes in the phonatory mechanism (Baken, 1997). Jitter was found to elevate (statistically and not statistically) in Parkinsonian subjects compared to controls in some studies (Gamboa et al., 1997; Hertrich & Ackermann, 1995; Holmes et al., 2000; Kent et al., 1994; Ramig et al., 1988; Zwirner et al., 1991). One study showed a decrease in jitter in the Parkinsonian patients but not statistical significance was reached (Ludlow et al., 1983). The statistically significant increased jitter measure was found in three studies (Gamboa et al., 1997; Hertrich & Ackermann, 1995; Zwirner et al., 1991). Holmes et al. report that increased jitter is attributed to the late stage of the disease while no differences were found in Parkinsonian subjects with early disease compared to controls. The latter conclusion seems logical in the light of studies that report an increased jitter and shimmer in aged voices (Linville, 2000) even though other factors such as the general state of health and fitness may also play a role (Colton & Casper, 1996; Linville, 2000; Ramig & Ringel, 1983).

A number of numerical indices of jitter have been proposed (see a relevant discussion by Baken, 1997). Absolute measures (perturbation factor and directional perturbation factor) and frequency related measures (period variability index, relative average perturbation, and deviation from linear trend)

have been used. Most of the studies that measure jitter in Parkinson's disease use the relative average perturbation measurement (Koike's algorithm) that is derived from the acoustic signal of the CSL speech software (Hertrich & Ackermann, 1995; Holmes et al., 2000; Gamboa et al., 1997). Differences in measurement of jitter in the present study compared to the aforementioned studies exist. In the present study a fundamental frequency cycle by cycle analysis is derived from the Lx waveform comparing directly the periodic structure of the wave (Howard, 1998) (see a relevant discussion of the rationale for the level of precision of this type of measurement in Titze, Horii, and Scherer, 1987).

Shimmer or intensity perturbation (summation of absolute differences in consecutive period amplitudes divided by the number of periods minus one), was found to elevate (not statistical significance) in a group of Parkinsonian patients (Gamboa et al., 1997; Holmes et al., 2000; Ramig et al., 1988) or in a female subgroup of Parkinsonian patients (statistical significance) compared to controls (Kent et al., 1994). Finally, one study found that increased shimmer was correlated with breathiness (Ludlow & Bassich, 1984). This finding probably reflects the bowing of the vocal folds. In hypokinetic dysarthria, the bowing of the vocal folds may result in "greater airflow turbulence and, therefore, more variation in intensity between periods" (Ludlow & Bassich, 1984, p. 187). The relationship between breathiness (perceptual measure) and shimmer was a valid procedure for intensity variation for speech (Ludlow & Bassich, 1984).

The measurements of shimmer are done in the same way as the measurements of jitter. However, in contrast to jitter, its data are obtained from the maximal peak-to-trough amplitudes of the individual waves. Shimmer

measurements include directional perturbation factor, amplitude variability index, shimmer in dB and amplitude perturbation quotient. Some of the studies in Parkinson's disease use shimmer in dB (Horii's algorithm) that is derived from the acoustic signal of the CSL speech software (Holmes et al., 2000; Gamboa et al., 1997). Alternatively, Ludlow and Bassich (1984) use the deviation from the linear trend in amplitude measurement which again is analogous to the same method from which jitter is derived. Shimmer in the present study was derived from the corresponding to the Lx signal speech signal using the Gold-Rabiner algorithm and the cepstrum analysis (Howard, 1998).

Perceptually determined, impairment of vocal loudness has been reported by Parkinsonian patients to be a frequent symptom of phonation (Duffy, 1995; Ludlow & Bassich, 1984; Schulz & Grant, 2000). Reduced loudness range (variability) is also a frequent perceptual feature (Gentil & Pollak, 1995). As a correlate to vocal loudness, average speech intensity was not found to be impaired in different studies (Canter, 1963; Ludlow & Bassich, 1984; Metter & Hanson, 1986). However, when Parkinsonian patients were asked to produce intensity in different levels (quiet, average, loud, and shout) during the production of the word [no], significant differences in mean intensity were found in the loud and shouted conditions (Canter, 1965a), and in the mean intensity that was driven from the final word of six sentences (Ludlow & Bassich, 1983). Finally, a reduced intensity range (difference between peak sound pressure level on shout production minus that on soft production in [a] and [no]) has been reported in patients with hypokinetic dysarthria (Ludlow & Bassich, 1983, 1984).

In reading and monologue, no differences were found in mean intensity (Canter, 1963; Holmes et al., 2000) and intensity range (Canter, 1963).

However, the standard deviation of intensity has been found to increase in controls as compared to Parkinsonian subjects (Gamboa et al., 1997) while no difference was found in early duration of disease Parkinsonian patients compared to late duration of disease patients (Holmes et al., 2000).

Special attention should be given to one fairly recent study that measured phonatory ability in Parkinsonian patients in early disease (mean duration 2.4 years), late disease (mean duration 13.2 years), and controls (Holmes et al., 2000). The authors reported that the progression of the disease is accompanied by the progression in severity of its phonatory features. No significant differences were found in the Parkinsonian group with early Parkinson's disease compared to controls. In contrast, speaking fundamental frequency in monologue, standard deviation of fundamental frequency, jitter and shimmer in sustained phonation were found to be significant in patients with late Parkinson's disease compared to controls. No information about matching in age between the groups is a major disadvantage of this study.

Fox and Ramig (1997) suggested that the sound pressure level (SPL) should be evaluated in a variety of tasks including maximum phonation time, reading, monologue, and picture description. Their results showed that SPL was significantly lower (2-4 dB SPL) in the Parkinsonian patients compared to controls in all tasks. Impaired laryngeal functioning of vocal fold adduction rather than of respiratory effort is probably the cause of the significant differences, which were found between the groups in SPL (Fox & Ramig, 1997). Electromyographic evidence supports the latter assumption. One study examined the activity of the thyroarytenoid muscle during sustained phonation, reading, and conversation (Baker, Ramig, Luschei, & Smith, 1998) in three

groups of subjects (one Parkinsonian and two controls). Under laryngostroboscopic examination, the Parkinsonian group exhibited reduced loudness, breathiness, and vocal fold incompetence. The electromyographic results showed that the Parkinsonian group exhibited the lowest amplitude of thyroarytenoid activity compared to the other groups. Although an acoustic analysis also took place, no significant results among groups were found. The authors supported the notion that these findings are associated with the neurological symptom of bradykinesia and the voice symptom of hypophonia. However, no information was given about probable motor fluctuations in the Parkinsonian subjects since the duration of the disease post onset was 4-5 years and the neurological stage in Hoehn and Yahr scale was 3-4.

Recently, other researchers examined the hypothesis that the reduced loudness of patients' speech and voice is attributed to a sensory processing deficit (Dromey & Adams, 2000; Ho, Iansek, & Bradshaw, 1999). In other words, when patients speak with a soft voice or reduced loudness it is because they perceive their voice to be at a normal level. The results of both studies showed no deficit in perception (at least at a severe level). Dromey and Adams (2000) hypothesised that the existence of no differences in their study may be caused by the insensitivity of their task (sustained phonation). Ho et al. (1999) hypothesised that the speech amplitude of Parkinsonian patients is preset lower due to a reduction of set production in basal ganglia. So, although their patients could regulate volume during different distances as well as their matched controls, their volume in conversation was lower than the controls. According to the authors, these results in voice volume mimic the symptoms of limb movement in Parkinson's disease (the relation of stride length and stepping rate

stays the same but the stride length is smaller).

The reported variability of speech and voice symptomatology in Parkinson's disease is the result of different instrumentation and tasks among studies and a differential selection of subjects (Kent et al., 1994). Factors such as duration of disease and neurological stage were not consistent in most of the studies (an exception to this fact was the study by Holmes et al., 2000). Kent et al. suggest that a careful choice of matching variables such as age and gender (between Parkinsonian groups and controls) and a similar neurological stage (in Parkinsonian groups) must be taken into account. The present study aims to investigate speech and voice in a homogeneous group of patients with an early diagnosis of Parkinson's disease (Hoehn and Yahr stage, I) matched in age, gender, and education with a normal control group of subjects. However, limiting factors such as differences between the Parkinsonian and the control groups in paralinguistic aspects (emotion), extralinguistic aspects (size and shape of laryngeal structures as well as physical health (Ramig & Ringel, 1983), and the lack of norms in the Greek population in fundamental frequency, fundamental frequency range, and intensity are limiting factors to the interpretation of the results of the present study.

1.5.4 Studies examining articulation

Even though laryngeal dysfunction in Parkinson's disease has an increased incidence compared to articulation (imprecise consonants), the latter is useful to describe the intelligibility of speech and the subsystem involvement (Kent, Kent, Duffy, & Weismer, 1998b). Because almost all of the studies that investigated articulation in Parkinson's disease included subjects with a

moderate to severe dysarthria and no control on duration of disease and neurological stage (mostly advanced neurological stage), the discussion on articulation will be limited to the most prominent studies.

In articulation, consonant imprecision has been the perceptual deviation reported most commonly (Canter, 1965b; Chenery et al., 1988; Darley et al., 1969a, 1969b, 1975; Logemann et al., 1978; Zwirner & Barnes, 1992). Logemann and Fisher (1981) studied 90 patients diagnosed with idiopathic and post encephalitic Parkinson's disease (at all five stages of the disease). Speech samples of the sentence version of the Fisher-Logemann Test of Articulation competence were recorded and analyzed by two trained phoneticians. Table 1 below shows the progression of misarticulations in each phoneme with the associated number of patients exhibiting them.

Table 1. Misarticulated phonemes in Parkinson's disease (taken from Logemann & Fisher, 1981).

Phonemes Misarticulated	Number of Patients
/k, g/	90
/k, g/ + /s, z/	63
/k, g/ + /s, z/ + /ʃ, ʒ /	43
/k, g/ + /s, z/ + /ʃ, ʒ/ + /tʃ, dʒ/	39
/k, g/ + /s, z/ + /ʃ, ʒ/ + /tʃ, dʒ/ + /p, b/	29
/k, g/ + /s, z/ + /ʃ, ʒ/ + /tʃ, dʒ/ + /p, b/ + /f, v/	21
/k, g/+ /s, z/ + /ʃ, ʒ/ + /tʃ, dʒ/ + /p, b/ + /t, d/	18

The authors emphasised that all of the misarticulations had a consistent pattern, which was characterised by incomplete closures for stops ([k] → [x]) and a partial but insufficient constriction of the vocal tract for fricatives ([s] → [s^{T}]) when T denotes lowering position of the tongue. This pattern represents an inadequate narrowing of the vocal tract at the point of articulation and it might be explained by the accelerated rate or reduced range of movement due to the neurogenic weakness of Parkinsonian patients (Canter, 1965b; Duffy, 1995; Logemann & Fisher, 1981). Gentil and Pollak (1995) explain the mechanism of the distortions in stop consonant production. While the normal production of a stop consonant involves a complete articulatory obstruction to the airstream, this is not the case in hypokinetic dysarthria. The complete obstruction is replaced by an incomplete obstruction resulting in the airflow to pass through a narrow constriction to generate a turbulent noise and a fricative-like production (spirantization).

The acoustic analyses supported the above findings (Ackermann & Ziegler, 1991; Kent & Rosenbek, 1982; Weismer, 1984a). A reduction of acoustic contrast or detail, indistinct syllable boundaries and spirantization were found in which stop gaps are replaced by low-intensity frication (Kent & Rosenbek, 1982; Weismer, 1984a). In addition, Weismer (1984a) discussed that the most important articulatoty feature of hypokinetic dysarthria is the shortened duration of voiceless fricatives measured from the glottal pulse preceding aperiodic energy to the first glottal pulse following the aperiodic waveform. However, no control of stage and duration of the disease took place in this study.

Ackermann and Ziegler (1991) examined the speech of 12 patients diagnosed with idiopathic Parkinson's disease during sentence imitation. Their

results were similar to the previous studies in stop consonant production. However, the phenomenon of spirantization was not uniform but was influenced by the specific linguistic demands (the intensity in the openings and closures that were associated with a stressed vowel and a post vowel consonant were performed at the expense of post vowel unstressed consonant). The rate between opening and closing movements of the articulators in sentence production seemed undisturbed.

Electromyographic studies (EMG) showed the same pattern of gradual decrease in velocity of lip displacement and a synchronous activation of agonist-antagonist muscles (anterior digastric and mentalis muscles for the production of [pa]) instead of a reciprocal action. This lack of reciprocity occurred only during the production of [pa] in fast rates and not in regular performance. The reduction in the range of movements is probably the cause of such findings (Hirose, Kiritani, Ushijima, Yoshioka, & Sawashima, 1981).

Gentil and Pollak (1995) report that the perceptual impression of imprecise consonants is a complicated entity and it involves (among others) a number of possible manifestations such as incomplete closure of stop consonants, imprecision in articulatory location, reduction of acoustic contrast and voicing of voiceless stops due to the reduced range of movement and the rigidity of the articulators. However, it seems to be an agreement in the perceptual and acoustic findings of the studies on articulation. An excessive difference in intensity (between stressed vowel vs. the following unstressed stop) in one study (Ackerman & Ziegler, 1991), the reports of spirantization (Kent & Rosenbek, 1982; Weismer, 1984a) and the perceptual impressions of Logemann and Fisher (1981) on stop consonant production show the same tendencies

(Kent et al., 1998b).

Logemann et al. (1978) hypothesised that the progression of voice and articulatory features in Parkinson's disease follows a consistent pattern. Early Parkinson's disease is accompanied by laryngeal abnormalities and followed by posterior tongue, anterior tongue, and labial abnormalities (severe Parkinson's disease). Adams (1997) challenges this notion. Differential effects of production can happen when 2 vocal tract structures are equally affected. A 50% increase in lips and vocal fold rigidity will have an important effect on vocal fold vibration but not at the same level in lip movement.

Canter (1965b) states that the involvement of tongue in articulatory errors was higher than the lips. There were a limited number of reports during the nineties that investigated the relation of strength and endurance of the tongue with the speech of Parkinsonian subjects (Solomon, Lorell, Robin, Rodnitzky, & Luschei, 1995; Solomon, Robin, & Luschei, 2000). The results were conflicting. The first study that employed mild to moderate Parkinsonian subjects (I and II grades in Hohn & Yahr scale), found that tongue strength was correlated negatively to disease severity and overall speech defectiveness. This simply means that a greater number of articulatory errors (perceptually determined) were associated with a weaker tongue. However, when the data of this study were combined with another study that investigated the same variables in subjects with a variable disease severity, no correlations were found between speech and non speech measures (Solomon et al., 2000). Even though the effect of medication seems to be the same in both studies (both studies involved medicated subjects) differences in the study design of the second study may account for these discrepancies. No correlations occurred between speech and

non speech measures according to neurological stage of the disease in the Parkinsonian subjects. A consistent manipulation of disease severity (neurological stage) and speech/non speech movements may result in different findings.

Connor and Abbs (1991) investigated task dependency in the duration and velocity of jaw movement (visually guided opening of the jaw, opening of the jaw in syllable and phrase production) in Parkinsonian and control subjects. Differential effects were found. Increased duration of jaw movement was found in the Parkinsonian subjects compared to controls in visually guided movements but the opposite occurred in the phrase length. Similarly, a decreased ratio of velocity to movement amplitude was found in the visually guided jaw movements while the opposite was found in the phrase production. Even though the subjects were under medication and the effect of medication is beneficial to jaw movement, these findings suggest differential impairments in putamen and globus pallidus of the basal ganglia where different anatomical regions (leg, arm and face) and different specialised subgroupings of cells evoke certain types of movement.

1.5.5 Studies examining rate

Even though perceptually determined speech rate abnormalities in Parkinson's disease are frequent, there seem to be variable (slow or fast) in different studies (Chenery et al., 1988; Ludlow & Bassich, 1983; Netsell, 1986; Scott et al., 1985; Zwirner & Barnes, 1992). Adams (1997) reports that only 10% of Parkinsonian patients exhibit rapid speech rates. Metter and Hanson (1986) found the variability in rate (slow and fast) in Parkinsonian patients to increase

with an increase in dysarthria severity. Ludlow and Bassich (1984) reported that 83% of their Parkinsonian subjects were perceived as having variable rate. This tendency was reported as the most severe speech difficulty of their subjects. A mismatch between the perceptual impressions and the results of acoustic studies in speech rate exists. It is possible that the perceptual results in articulatory rate in Parkinson's disease may overestimate the actual rate that is based on physical measures (Tjaden, 2000).

Weismer (1984a) suggested that the perception of fast rate might be due to the articulatory imprecision and continuous voicing that reduces acoustic contrasts and results in a "blurring" of speech. Other studies suggest that the perception of accelerated speech or fast rate might be the result not in increases of the rate of articulatory movements but in the reduced range of movements (Kent & Rosenbek, 1982; Tjaden, 2000).

Many studies have found the additional feature of "short rushes of speech" perceived as an "accelerated" speech pattern (Chenery et al., 1988; Darley et al., 1975; Scott et al., 1985; Zwirner & Barnes, 1992) and considered it as one of the most prominent features of hypokinetic dysarthria. Its manifestations are apparent in Alternating Motion Rates (AMRs) or diadochokinetic rates in syllable production ([pa] [ta] [ka]). AMRs have been used to evaluate the speed and regularity of jaw, lips, and tongue. Rapid or accelerated rate in AMRs shows the speech rate abnormalities. Canter (1965b) has found significant differences in the Parkinsonian subjects compared to controls in AMRs. Other studies found no rate differences in AMRs or syllable repetition tasks (Connor, Ludlow, & Schulz, 1989; Ludlow, Connor, & Bassich, 1987). Hirose et al. (1981) using physiological methods, found a tendency for

increased rate and reduced range of lower lip movement on syllable repetition tasks. However, their results were descriptive in nature since no group comparisons were attempted (only 2 Parkinsonian subjects were employed).

One study suggested that Alternating Motion Rates (AMRs) might be sensitive measures to identify speech problems in the preclinical period of Parkinson's disease (Parnell & Amerman, 1996). AMRs were investigated longitudinally in normal elderly subjects in an 11 year follow-up. One subject who took part in the study was diagnosed with Parkinson's disease 5 years after the first speech assessment. His AMR scores were measured acoustically, and separately from the group of his peers in the first and second speech assessment (after 11 years). The results showed that the rate of variability in AMRs (average period difference between 20 consecutive movement cycles in [ta] divided by the average vowel-to-vowel period and multiplied by 100) was higher in the Parkinsonian subject 5 years before the neurological diagnosis (first speech assessment). In the first speech assessment, the non speech volitional control of the Parkinsonian subject in most tongue movements was found to be slow. The authors suggest that AMRs may be sensitive indicators of changes in the central nervous system in tasks of maximum oral performance which are not however necessarily perceptible.

Acoustic analyses have shown an intersubject variability in speech rate (Canter, 1963; Hammen & Yorkston, 1996). No statistically significant differences were found. Canter (1963) reported differences in some of his subjects, with some subjects exhibiting slow rates, and other subjects exhibiting extremely fast rates. Hammen and Yorkston (1996) reported longer pauses in the habitual speech in their subjects, an increased percentage of pauses to

occur within clause/phrases ('inappropriate pauses'), and shorter speech duration (excluding pause duration). Ludlow and Bassich (1983) found the Parkinsonian subjects to exhibit a slower rate in fast sentence production than the controls. Ludlow et al. (1987) did not find sentence production rates abnormally fast. In general, the Parkinsonian group did not differ from the control group in the speech rate but instead in the change of speech rate from regular to fast during sentences and phrases. So, the Parkinsonian subjects exhibited a reduced ability to alter speech rate. In particular, what distinguished the Parkinsonian group from the normal control group was a change in sentence duration (duration difference between a regular minus a fast rate in production of a sentence) (Ludlow & Bassich, 1984; Ludlow et al., 1987). In addition, a percent reduction in phrase duration was found as a difference in milliseconds between regular minus fast time divided by the regular time and multiplied by 100 (Ludlow et al., 1987). During longer and more complex utterances (sentences and phrases) compared to syllables (no difference was found), a reduced percent duration change was manifested in the Parkinsonian group. The authors concluded that the basal ganglia disease seems to affect the rate of speech movements and their controlled alteration during speech execution but not during initiation or planning. However, Kent et al. (1998b) report that the above findings are not unique in hypokinetic dysarthria of Parkinson's disease but occur in other dysarthrias.

1.5.6 Studies examining consistencies between perception and acoustics

The great variability of the results of perceptual research on the laryngeal system not only in Parkinsonism and idiopathic Parkinson's disease but also in

other neurological disorders have often been discussed (Leuschel & Docherty, 2000). This is a problem of perceptual judgement and some authors have pointed it out. Fex (1992) considered whether the great variability of characteristics found on perceptual assessment may be due to the abstractness of definitions and to different opinions from listener to listener based on individuality of reference levels in voice quality. According to Baken (1997) perceptual judgements alone may mislead the clinician due to factors such as unreliability of listeners' judgement of pitch and interaction in a complex way of frequency, intensity and spectral properties to determine pitch perception. For example, the perceived abnormality of pitch may reflect the speaker's vocal intensity. In contrast, the instrumental methods of voice measurement enable the examiner to isolate specific parameters for measurement, but they still cannot replace the human ear (Baken, 1997). An ideal instrumental or acoustic analysis will be able to isolate parameters from the signal and relate them to physiological factors and perceived voice quality (Wirz & Beck, 1995). One solution to this problem could be if the clinicians/researchers were trained through acoustic recordings, and if high quality tape recordings could be used for assessment based on sustained phonation and running speech samples with well-defined noise components (Fex, 1992).

In dysarthria, a few studies have found consistencies between perception and acoustics. This may create a problem especially in the present study in which the subjects are at the beginning of Parkinson's disease and, at least perceptually, are predicted to be close to normal. Baken (1997) concludes that "Comparison to a norm requires that both the norm and the behaviour in question be based on unbiased and objective scales..." (p. 125-126). The lack of

norms in the speaking fundamental frequency in the Greek language further complicates the problem and limits the interpretation of the results in the present study.

The examination of perceptual ratings and acoustic measures of hypokinetic speech to find the most appropriate measures for assessment has been carried out by Ludlow and Bassich (1984) in 12 patients diagnosed with idiopathic Parkinson's disease and the same number of controls matched by age and gender. The speech samples were perceptually and acoustically analysed (from spectrograms). The most important acoustic measures that were related to perceptual measures included factors that reflected laryngeal control (from most severe to less severe):

- The number of on-off phonations on vowel [a] repetition (the number of vowels produced in 5 seconds minus the number of gaps in phonation in 5 seconds)
- The number of consonant voicing errors (the number of syllable repetitions produced in 5 seconds minus the number of gaps in phonation in 5 seconds)
- The total range in fundamental frequency on a pitch glide task (the difference in Hz, between low and high points of an ascending vowel production divided by mean fundamental frequency in sentences) and,
- The range in intensity between soft and shouting voice during the production of [a] and the word [no] (difference between peak sound pressure level on shouting production and that on soft production).

In conclusion, acoustic measurements in this study indicate an inability of the Parkinsonian patients to manoeuvre speech range in fundamental frequency and intensity. Correlation coefficients between Fo range and monopitch on the

one hand and intensity range and monoloudness on the other were high (.65 and .67 respectively). This fact, points to the possibility that patients with Parkinson's disease are unable to use their larynges and articulators in order to provide the prosodic aspects of speech, and that this may be caused by rigidity. Zwirner et al. (1991) found a relation of the severity of dysphonia, and the acoustic measure of standard deviation of fundamental frequency (SDFo) during sustained phonation in patients with Parkinson's disease.

One possible reason that not many studies have found consistencies between perceptual and acoustic findings is the use of tasks that require maximum performance. Many of the studies that measured phonation and speech rate in Parkinson's disease have used maximum performance tests (maximum phonation duration, fundamental frequency range, maximum expiratory pressure, maximum sound pressure level, and maximum diadochokinetic rate) (Canter, 1963, 1965a, 1965b; Connor et al., 1989; Fox & Ramig, 1997; Hirose et al., 1981; Ludlow & Bassich, 1983, 1984; Ludlow et al., 1987; Parnell & Amerman, 1996; Solomon et al., 1995, 2000). These tests examine the upper limits of performance for selected speech tasks (Kent, Kent, and Rosenbek, 1987). Although the application of such tests in motor speech disorders seems reasonable because of their similarity to tests that are used in clinical neurology and measure strength, range, or speed (Kent et al., 1987) they also have disadvantages which can confound the results. Kent et al. (1987) review the merits and drawbacks of maximum performance tests.

Their advantages involve their frequent use in screening and assessment of motor speech disorders, their potential for international use since they do not require a specific language, and their ability to fit to the demands of ordinary

speech production, which falls well within their limits (Kent et al., 1987; Kent & Kent, 2000). Non speech tasks of the oral mechanism may also provide information about the existence of motor speech disorders including Parkinsonism by assessing the interaction between the motor and linguistic contributions and by separating the motor processes and the linguistic levels of constraint (Robin, Solomon, Moon, & Folkins, 1997). Finally, they are helpful in assessing the relative contribution of a given speech production subsystem (respiratory, phonatory, velar, and articulatory) to the disorder (Robin et al., 1997).

However, a number of disadvantages make them suitable for use only in conjunction with other tasks, e.g. reading and conversation. The major disadvantages of these tests involve (Kent et al., 1987):

- The lack of standardised procedures for their application
- The large intrajudge and interjudge variability in the performance of subjects due to the effect of practice, motivation, inappropriate instructions, differences in age and sex and other physical variables among subjects.
- The lack of normative data, especially in view of life-span considerations.
- The non established relationship to speech. Each of the maximum performance tests is a non speech behaviour that has a presumed relationship to speech or a stylised speech activity such as the prolongation of a vowel.

Kent et al. (1987) conclude that "properly used, the maximum performance measures may be useful and justifiable...but the problems outweigh the suggested solutions in some, and perhaps many, of the areas of concern." (p. 384). In contrast, Robin et al. (1997) support the notion that despite their

weaknesses the combined use of speech and non speech tasks may be beneficial for the assessment of the speech motor mechanism.

In the present study these tasks were considered useful to show possible motor abnormalities since the Parkinsonian subjects were at the beginning of the disease and it was predicted to be close to normal. The use of maximum performance tasks in the present study took place through the Frenchay dysarthria assessment, the advantages and disadvantages of which will be discussed in section 2.5.

1.5.7 Other studies

The term prosody refers to linguistic and non linguistic information and involves intonation, sentence accent, word stress, and rhythm of speech. The perception of prosody relies, into a large extent, on fundamental frequency, intensity, and syllabic duration. All of these aspects have been discussed separately in the previous sections. This section will deal with studies on dysarthria that focus on stress and intonation and studies in the Greek language of normal speech production since there is a lack of Greek studies in dysarthric speech. Also, an additional study will be considered because it used different methodology from the aforementioned studies even though this study does not deal with stress and intonation.

Prosodic disturbances in the form of reduced stress exist in most of the perceptual studies (Chenery et al., 1988; Darley et al., 1969a, 1969b, 1975). Ludlow and Bassich (1984) found that reduced stress was correlated with the acoustic measure of interword interval length. This measure was defined as a difference in time between two words with equal stress ("blue bell) and one

compound noun ("bluebell") when produced in same sentences. In this way, duration changes in time to mark word barriers were found. The Parkinsonian patients exhibited shorter intervals than normal controls.

Stress and intonation have been examined acoustically (Hertrich & Ackermann, 1993; Kent & Rosenbek, 1982; LeDorze, Ryalls, Brassard, Boulanger, & Ratté, 1998; Ludlow & Bassich, 1983; Murry, 1983; Penner, Miller, Hertrich, Ackermann, & Schumm, 2001). All studies are characterised by different ways to assess stress and intonation even though they used acoustics as a method of instrumentation. Kent and Rosenbek (1982) found an overall reduction in acoustic contrast or detail and "a flattening of the acoustic relief across the syllable chain" (p. 282). In sentences with linguistic meaning, Ludlow and Bassich (1983) found a severely restricted range of fundamental frequency during stress contrasts and during imitation of sentence fundamental frequency contours. Murry (1983) found increases in frequency and intensity during effort to stress a word in initial and final position. Hertrich and Ackermann (1993) found a slightly decreased duration of the stressed syllable in Parkinsonian subjects exhibiting dysarthria.

Intonation (difference in mean fundamental frequency on the last syllable of the same sentence produced as both question and as a statement) was found to be lower in the Parkinsonian group compared to controls (Le Dorze et al., 1998). Another study investigated intonation by examining pitch accent patterns instead of variability in one sentence produced with three ways: descriptive, angry, and interrogative (Penner et al., 2001). The Parkinsonian subjects exhibited a tendency toward a lower rise in relative fundamental frequency than the controls. However, a high performance variability between the subjects, their

small number (N = 3), and the different duration of the disease (9-14 years) were major disadvantages in this study.

Variation in the voice source may be associated not only with segmental elements of a language (contrastive use of voice quality for vowels or consonants in some Asian, African and American languages) but also with suprasegmental elements of a language (relation of fundamental frequency to intonation, tone and stress). Individual differences in voice quality may be the result of a number of factors involving the physical properties of the vocal apparatus and linguistic, paralinguistic and sociolinguistic influences (Chasaide & Gobl, 1997). Any or all of these factors may signal variation in the voice source and show differences in numerical indexes such as fundamental frequency and intensity.

Although the present study aims to identify phonatory differences between Greek Parkinsonian subjects and controls, its assumptions were based on studies which focused on English speaking populations. Linguistic differences between Greek and English may be important factors in how pathological voice is used by the Parkinsonian subjects. A significant limitation in the interpretation of the results of the present study is the differences between stress and intonation in the Greek language and the English language. Stress is an important factor that differs among various languages. In normal speech production of the Greek language, research on these aspects starts to unveil its differences compared to other languages.

In normal speech production of English rhythm, there is an even alternating of stressed and unstressed syllables, while in Greek this is not the case. In the Greek language a requirement for alternation of strong and weak

syllables does not exist since there is no elimination of stress lapses that is observed in English and other stress-timed languages (Arvaniti, 1994). Irregularity and sparsity of stresses and relatively stable duration of syllables (with the exception of syllables with primary stress) are characteristics of the Greek language similar to the Italian language (Arvaniti, 1994). The resolution of clashes is achieved by different ways in syllable-timed languages (such as the Greek language) compared to stress-timed languages (such as the English language). In the Greek language distressing or inserting extra duration between clashing syllables is the primary strategy while in English shifting stress results in more evenly alternating prominent syllables (Arvaniti, 1994).

Other characteristics of lexical stress in the Greek language involve the existence of Stress Well Formedness Condition (SWFC) which allows stress to occur on any of the last three syllables of a word and no further to the left. However, with the addition of an enclitic to a host stressed on the antepenultimate a stress is added two syllables to the right of the lexical stress (Arvaniti, 1992). The SWFC induced stress becomes the most prominent in the group and it is not perceptually different from the lexical stress. Arvaniti (1992) suggests that Greek may not have rhythmic stresses at all. Finally, an important characteristic of Greek stress is that the Fo high in the Fo contour is associated with the end of the stressed syllable and the beginning of the following and not with the beginning of the stressed syllable (Botinis, 1989; Arvaniti, 1992).

On the same lines, linguistic research in intonation in Greek shows that the H target (max turning point in an Fo contour) is very precisely aligned just after the beginning of the first post accentual vowel and it is very stable relative to the onset of the first post accentual vowel (independently of differences such

as the combined duration of the accented syllable and the immediately following consonant) (Arvaniti, Ladd, & Mennen, 1998). So, the Greek prenuclear accents are represented as bitonal accents of the form L (min turning point in an fo contour) and H (max turning point in an fo contour). The authors conclude that the "L and the H of the accent are anchored to segmentally defined positions, and the duration and slope of the pitch movement are completely determined by the segmental composition of the accented word" (p. 24).

The description of the aforementioned features is beyond the scope of the present study but they have been mentioned in order to show that their existence limits the interpretation of the results of the present study and prohibits their generalisation. For example, due to differences in stress and intonation, dysarthria in Parkinson's disease in the Greek language may show different compensations compared to the English language. Although, the present study focused on phonatory aspects only and stress and intonation were not measured, the complex relationship between fundamental frequency and intensity during reading and monologue may unveil the differences between languages and limit the interpretation of the results.

Finally, one study that used different methodology from the other studies will be described. This study used a different way to identify differences in Parkinsonian levodopa treated subjects. Metter and Hanson (1986) investigated brain anatomy and glucose metabolism with the speech characteristics in reading of 8 Parkinsonian patients and 2 patients diagnosed with supranuclear palsy and having variable degrees of hypokinetic dysarthria. Ten control subjects were matched to the experimental group by age. The acoustic measures of speaking rate, mean fundamental frequency, relative intensity, and vowel

phonation time were taken from tape-recorded speech samples and were compared to age-matched samples of controls. The results showed that there was a trend of increase in Fo with increased clinical disability and with increased severity of dysarthria. However, the mean fundamental frequency (Fo) for each patient was within the normal range compared to controls.

1.6 Electrolaryngography as a technique of voice measurement

Electrolaryngography (ELG) or electroglottography[1] (EGG) has been used in the last two decades as a clinical and assessment tool in the field of speech and voice pathology and therapy. Its basic aim is to monitor vocal fold closure and to record changes of vocal fold vibration during voiced sounds (Abberton, Howard, & Fourcin, 1989). Qualitatively, electrolaryngographic analysis can be used to complement auditory voice quality assessment including vocal registers and intonation analysis. On a quantitative basis, it can be used to measure physical and statistical characteristics of phonation and fundamental frequency. In addition, by analysing the vocal fold vibration (mode and rate), electrolaryngography relates them to the perception of voice quality and intonation (Abberton et al., 1989). Knowledge of basic physical measurements of voice can be associated with different disorders that affect voice. The extensive use of other instrumental methods in speech pathology, such as acoustics, is based upon this logic. In the end, change in these physical measurements with speech therapy may improve voice. In a sense, quantification could provide the stable ground for monitoring therapy.

[1] Fourcin (1981) uses the name electrolaryngography while other reseachers prefer the name electroglottography (Baken, 1992; Titze, 1990). Baken (1992) commented that the term electroglottography does not really represent its actual use because the signal does not give information about the glottis and its size but instead provides information about the closed phase of the vocal fold vibratory cycle. To avoid confusion, both terms will be used in this study reflecting the individual view of each researcher.

The electrolaryngograph measures conductance by using 2 electrodes, one on each side of the larynx at the level of the thyroid alae, and by passing a current between them during the production of voiced sounds. Its underlying purpose is to measure the electrical impedance (or conductance) during the opening and closing phases of the vibrating vocal folds. Air is an extremely poor conductor while tissue is a moderately good conductor. During the glottal cycle the electrical impedance in the larynx is increased when the glottis is open (air is between the vocal folds) and decreased when the glottis gradually closes. Under normal conditions impedance will show the details of laryngeal function (Baken, 1992, 1997; Colton & Conture, 1990; Kitzing, 1990).

Baken (1992) describes the function of a 'generic' electrolaryngograph. Figure 6 is taken from Baken and it has been modified for the purpose of this study. It shows a block diagram of a typical electrolaryngograph.

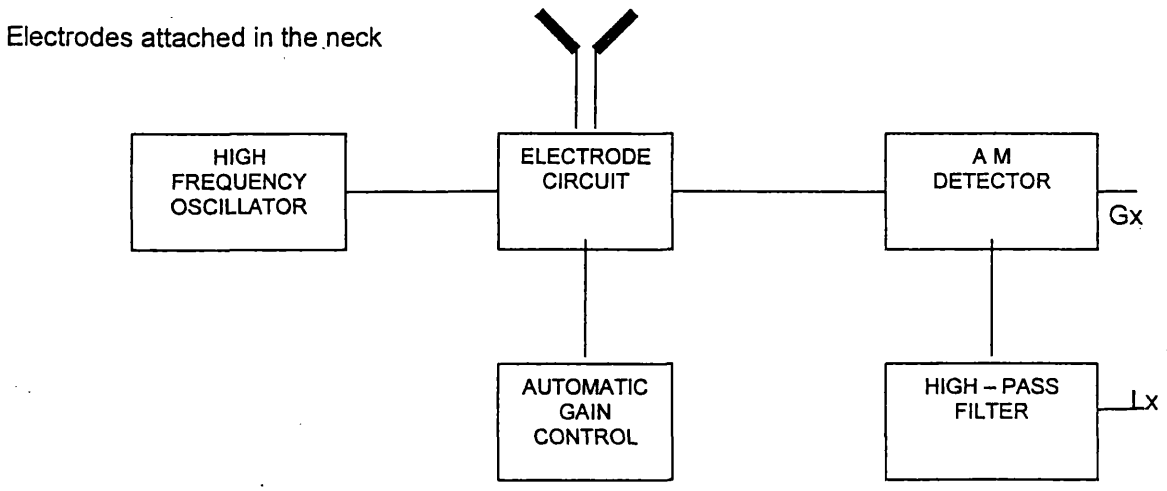


Figure 6. Block diagram of a typical electrolaryngograph (taken from Baken, 1992).

The oscillator generates a high frequency current (300 kHz - few megahertz). This current is coupled to the neck through the use of the electrodes. In modern electrolaryngographs, a guard ring that is attached to the

electrodes shields the electrodes from noise and restricts the spread of the electrical current in the neck. The voltage that is generated across the neck is 0.5 V. The high frequency current varies with changing impedance due to vocal fold movement. This results in a voltage drop between the electrodes. The decoding of this signal with the elimination of the high frequency waves produces waves that involve only their amplitude. This amplitude signal represents trans-neck impedance over time. The automatic gain control is used to change the amplitude of the carrier wave or the sensitivity of the detector system. In this way, a stable and large output will be obtained. The automatic gain control is also used to reduce a slow drift of the output baseline that is due to changes in the characteristics of the electrode-skin interface that occur over time. The high-pass filtering eliminates slow changes due to perilaryngeal activity and it passes rapid impedance variations for further amplification. As a result, a change in the amplitude of the carrier current occurs and an amplified clear signal appears (Lx). This signal shows only the glottal behaviour that is taking place during phonation, in contrast to another signal (Gx) that shows the large changes in impedance before and after the phonatory event (including the perilaryngeal activity).

The electrolaryngographic excitation output waveform (Lx) is the output waveform that has peaks when there is a contact of vocal folds (minimal impedance) and troughs when there is no contact (maximal impedance) and it gives the current flow as a function of time. The vibration of the vocal folds during voiced sounds results in a quasi-periodic Lx waveform with the continuous opening and closing of the vocal folds. More specifically, the start of each steep rise in the Lx output is associated with the onset of vocal fold closure,

the peak of each cycle is associated with maximal closure (horizontal and vertical) and the trough is associated with vocal fold separation (Abberton et al., 1989).

1.6.1 Relationship of electrolaryngography to vocal fold contact

Fourcin (1981) describes the details of the contact of the vocal folds on an Lx waveform. According to him, the first phase of the Lx signal involves the production of a mucus bridge between the approximating epithelial cover surfaces. This is the end of the open phase of vocal fold vibration and it is associated with contact between the covers at the lower edges of the glottis. The mucus bridge gives a very rapid jump up in electrical conductance across the neck. After this, the maximum electrical contact is associated with the snapping together of the covers of the two folds. An illustrated diagram (taken from Baken, 1997) shows the above demarcations of the Lx signal (Figure 7).

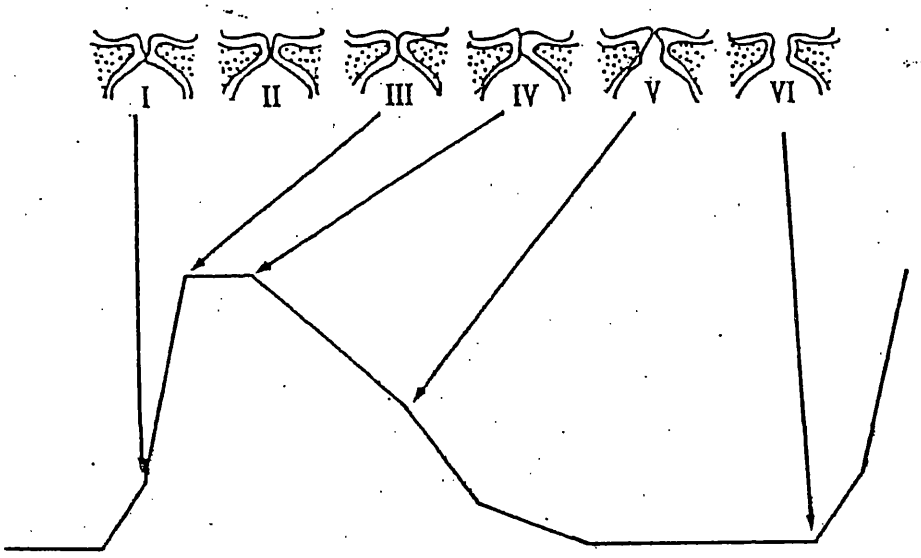


Figure 7. Electrolaryngographic views and corresponding positions of the vocal folds (Baken, 1997 based on MacCurtain & Fourcin, 1982).

Fourcin (1981) emphasises the importance of 5 points in the examination of Lx waveforms in normal voice:

- Uniform Lx peaks are associated with a uniform acoustic output.
- A sharp Lx contact is associated with a good acoustic excitation of the vocal tract.
- Long closure duration is associated with undamped formants.
- Regular sharply defined contact periodicity is associated with well defined pitch.
- A progressive change in sharply defined Lx period lengths is associated with a changing voice pitch.

Long samples (more than 1 minute) of speech are necessary in the ELG analysis. The reason is that in speech samples of more than one minutes there is a possibility that production difficulties (pathological voice) will arise more clearly. Even though the nature of the sample may affect the analysis, it is clear that a close to monotone or a monotone voice (as in Parkinson's disease) will probably give a narrower fundamental frequency distribution (Abberton & Fourcin, 1984).

Carlson (1995) states that Fourcin, compared to other researchers, modified the phase of the displayed output waveform in ELG. So, the Lx waveform is positive (upward) when there is an increase in vocal fold contact and negative (downward) when there is a decrease in vocal fold contact. A left tilt in the Lx waveform denotes the abruptness of closure compared to the gradual opening phase. As was stated above, the steepness of the slope of the Lx output waveform for the closing phase of the vocal folds also determines the acoustic excitation of the vocal tract. Finally, a simultaneously recorded acoustic

speech pressure waveform (Sp) can also be displayed and this denotes the output waveform of the microphone.

1.6.2 Quantitative measures of electrolaryngography

The modern use of electrolaryngography can provide quantitative measures of voice. These measures have been discussed extensively in many articles (Abberton et al., 1989; Abberton & Fourcin, 1984, 1997; Fourcin, Abberton, Miller, & Howells, 1995; Fourcin, 2000). By measuring the time during vocal fold closures, the researcher is able to find the fundamental period of excitation (Tx) and to derive the fundamental frequency of excitation (Fx). Based on Lx, many other voice parameters can be measured. Fundamental frequency changes on a period-by-period basis (Fx) are correlated with the perception of intonation or pitch patterns.

In general, Dx (the distribution of excitation fundamental frequencies on a probability histogram) during voiced speech gives a measure of vocal fold closure regularity and, in particular, a measure of frequency regularity and range. Intensity regularity and intensity range can also be displayed. Dx is presented in 2 orders (first order and second order). The first order gives the raw Tx data while the second order includes pairs of laryngeal periods that occurred twice in the same statistical 'bin'. Dx is plotted on a logarithmic scale corresponding to perceived pitch. Its frequency axis is divided into 128 logarithmically equal spaced 'bins' having an overall range from 30.52-1000 Hz. Each bin is a few Hz, in width (Abberton et al., 1989). In addition to Dx, a scatterplot (Cx) of each Fx (every measured Fo value) against the one that follows can also be used. Cx plot is comparable to the first order Dx plot. In Speech Studio (the computer program

that is used for the quantification of Fx measurements) the Tx values in the second order distribution are admitted based on a cycle-to-cycle control of vocal fold vibration. In this way, a large number of samples are included.

Both Dx orders, but especially the second order, represent vocal fold regularity (Abberton et al., 1989; Fourcin, 1981, 2000). Theoretically, the second order distribution is preferable to the first order because it does not include data, irrelevant to voicing, that might affect the calculations of Fx parameters (swallowing or excessive gross laryngeal movement during recording). However, the irregularities that are discarded by the second order may be a part of the voice. Because the present study measures fundamental frequency in a sample of experimental subjects that are predicted to be close to normal (at least neurologically with no serious decline of performance due to early beginning of the disease), both orders are used to examine if differences in the results between them exist.

Qx is defined as the mean percentage of time the vocal folds are closed (measured 70% down from the waveform peak) to the total period and it is related to the quality of excitation of the vocal tract as the vocal folds vibrate (Abberton & Fourcin, 1997; Fourcin, 2000; Fourcin et al., 1995). It is derived from the Lx waveform by determining the period of time between closing and opening of the vocal folds.

In summary, most of the measures that can be derived from electrolaryngography include:

- Measures of central tendency of fundamental frequency
- Fundamental frequency range
- Probability density functions of the duration of laryngeal silence

corresponding to voiceless portions and silence

- Durations of continuous larynx activity corresponding to voiced portions of speech
- Measures of vocal fold closed and open phases in each vibratory cycle and
- A phonetogram (range of intensities against their vocal fold frequencies).

1.6.3 Electrolaryngography in the analysis of voice in neurological disorders

In voice measurement of different disorders, many researchers have reported the use of electrolaryngography (ELG) alone or together with other techniques (Gerratt, Hanson, & Berke, 1987, 1988; Hanson, Gerratt, & Ward, 1983; Horiguchi, Tomoyuki, Baer, & Gould, 1987; Jiang, Lin, Wang, & Hanson, 1999). Fourcin (1981) states that Lx is important in voice assessment since events that occur when the vocal folds come into contact depend on the physiological condition of the vocal folds. So, different pathologies are associated with different contact phenomena. In a sense, quantitative measures of phonation depend on vocal fold contact. Because the studies that used ELG in neurological disorders are limited, an extensive description of them will follow.

In the eighties, Hanson, Gerratt, and his colleagues (Gerratt et al., 1987, 1988; Hanson et al., 1983) emerged as advocates of measuring voice abnormalities in neuromuscular disorders. They suggested the use of a combined method of phottoglottography (PGG) and electroglottography (EGG) to measure laryngeal structure and function. PGG was suggested to provide information about the open glottis while EGG was suggested to describe glottal contact during the closing phase. The authors reported that the combination of

both methods can give information about the opening and closing phases of vocal fold vibration and the use of measures such as speed quotient (SQ) and open quotient (OQ) could be beneficial to accomplish this task. Speed quotient was defined as the ratio of the duration of the lateral excursion of the vocal folds (in ms) divided by the duration of medial excursion of the vocal folds (in ms). The authors also suggested that the speed quotient measure might be related to myoelasticity. Open quotient was defined as the ratio of the duration when the glottis is open divided by the total duration of the glottal cycle. According to them, the visual aspect of the EGG signal makes it an attractive method of vocal assessment. In addition, EGG offers insights into the more subtle vibratory and tension abnormalities that are associated with abnormal phonation. The authors stated that "information obtained from glottographic studies is often diagnostically helpful, particularly in patients with voice abnormalities associated with neuromuscular disorders" (Hanson et al., 1983, p. 418).

Hanson et al. (1983) compared sustaining [i] sound measurements of voice in 3 subjects with different voice pathology (one Parkinsonian subject, one subject diagnosed with spastic dysphonia and one subject poisoned by arsenic) to a control subject of the same sex. The Parkinsonian subject was found to have an increased fundamental frequency mean compared to the subject with the normal voice (PD Fo mean = 119 Hz, PD OQ = 0.84, Normal Fo mean = 106 Hz, Normal OQ = 0.44). The EGG waveform of the Parkinsonian subject showed a remarkably longer time for the opening portion of the glottal cycle (71% of the entire cycle) and a significant reduced closing portion of the cycle (15% of the entire cycle was spent "in the most closed period"). The waveform shape also varied from cycle to cycle indicating a marked variability of vocal fold posture.

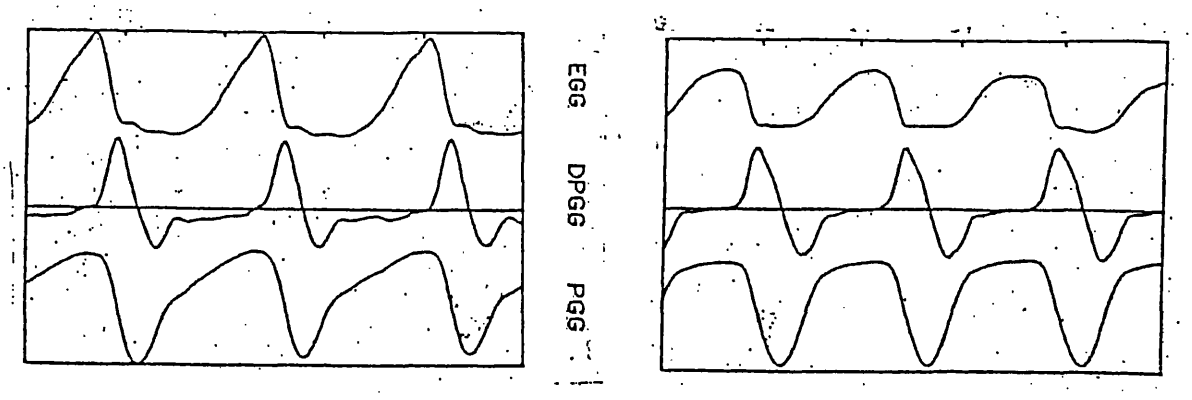
The physiological interpretation of the results was that the vocal folds of the Parkinsonian subject during phonation moved toward the midline but did not make complete contact at their medial edges. As a result, the rising and falling slopes lacked the continuities associated with the medial contact of the upper and lower vocal fold margins that should occur during normal phonation. So, the vocal folds of the Parkinsonian patient looked bowed during phonation. Furthermore, the glottic gap was visibly greater during phonation correlating with a breathy voice production, short phrasing of speech, and decreased ability to sustain phonation. All of these findings were associated with the laryngoscopic examination. The authors emphasised the importance of glottographic waveforms to provide diagnostic information such as abnormally increased vocal fold tension or cycle-to-cycle variability in vibration.

Using the same technique, the authors analyzed recordings of sustained [i] in subjects with reported damage to the nervous system that in turn affected their phonatory ability (Gerratt et al., 1987). Emphasis on these aspects could enhance the ability to understand the normal motor control of laryngeal function. Four subjects (one male with recurrent nerve paralysis, one male with superior laryngeal nerve paralysis, one male with Parkinson's disease, and one normal control male) were examined. The Parkinsonian patient, as compared to the normal control subject, exhibited increased fundamental frequency, speed quotient, frequency perturbation ratio (jitter ratio), and amplitude fluctuation ratio (shimmer ratio). Table 2 is taken from Gerratt et al. and shows the measurements of all subjects in this study.

Table 2. Voice measurements in a normal control subject, a Parkinsonian subject and two subjects with laryngeal nerve paralysis.

SUBJECTS	Speed Quotient In ms	Fo in Hz	Frequency Perturbation Ratio (jitter ratio)	Amplitude Fluctuation Ratio (Shimmer ratio)
Normal Control Subject	0.98	110	8.64	29.80
Parkinsonian Subject	2.90	118	9.74	39.90
Subject with Superior Laryng. Nerve Paralysis	1.28	187	18.46	107.70
Subject with Recurrent Nerve Paralysis	0.35	154	8.15	33.40

Figure 8 shows illustratively the waveforms of the Parkinsonian subject vs. the normal control subject. For the sake of the current discussion the lower diagram was reversed to represent the electroglottographic signal with the closing phase represented at the top of the signal (upward). DPGG is the change of acceleration of the vocal folds from opening to closing.



Fo = 118 Hz
SQ = 2.9
OQ = 1.0

Fo = 110 Hz
SQ = 0.98
OQ = 0.60

Figure 8. Photoglottographic and electroglottographic signals of a patient with Parkinson's disease (left) and a person with normal phonation (right).

Supported evidence came from other studies that used electromyography. Bizarre high frequency discharges and big potentials during phonation were found in two out of eight Parkinsonian patients in the region of posterior cricoarytenoid muscles and in the interarytenoid respectively (Guidi, Bannister, & Gibson, 1981) and in the region of the intrinsic laryngeal muscles (Hirose, Sawashima, & Niimi, 1988). Gerratt et al. (1987) explained that this activity could cause a greater than normal resistance to the opening of the vocal folds since subglottal pressure increase during phonation (slower opening of the vocal folds and a long open phase). When the vocal folds travel to the limits of the excursion, increased myoelasticity could cause the folds to move back to the midline quickly. In this study, an increased SQ and stiffness of the vocal folds in the Parkinsonian patient and a decreased SQ in the patient with unilateral lower motor neuron paralysis were found. Murdoch, Manning, Theodoros, and Thompson (1995a) did not confirm the above results. The authors found no differences between control subjects and 20 Parkinsonian subjects in fundamental frequency and closing time even though the perceptual assessment revealed that 89.5% of the Parkinsonian subjects exhibited laryngeal features such as breathiness, glottal fry, hoarseness, and pitch unsteadiness.

Except from quantitative measures, electrolaryngography has also been used clinically to observe traces of dysphonias in 432 patients (Motta, Cesari, Iengo, & Motta, 1990). Of the 432 patients, 50 were not affected by dysphonia, 66 presented hypokinetic dysphonia, 85 exhibited hyperkinetic dysphonia, 92 had nodules in one or both vocal folds, 86 had polyps of vocal folds, and 53 exhibited Reinke's edema. The results showed the appearance of different traces characteristic of the disorder in the Lx record. The glottal wave from

patients with hypokinetic dysphonia showed a sharp peak and reduced amplitude in almost all of the patients (93% of cases).

There is only one study that measured the effect of pharmacology on voice (Jiang et al., 1999). The authors found that acoustic and electroglottographic signals were useful in monitoring the pharmacological response of Parkinson's disease on voice parameters. The speed quotient that was obtained from EGG signals was analysed to detect change in vocal fold rigidity while jitter and shimmer were obtained to detect change in phonatory instability. Ideally, in normal subjects speed quotient equals 1. The results of EGG vs. acoustic measures showed high correlation ($r = 0.99$, $N = 30$) in fundamental frequency. Although there was a great variability between subjects, and across speech performance, the findings of EGG supported the notion that in some subjects medicated with levodopa the speed quotient decreased. This finding may also suggest that the increased speed quotient measured from EGG signals in Parkinsonian patients before treatment may be related to muscle rigidity, thereby causing increased vocal fold stiffness. In the same study, other results showed that levodopa treatment increased acoustic SPL, a conclusion that suggests that levodopa may improve either vocal efficiency through increasing respiratory volume or amplitude of vocal fold vibration, or both.

1.6.4 Frequency perturbation and amplitude perturbation as measures of voice quality in electrolaryngography

As it was indicated in previous sections, jitter and shimmer are measures that can be related to the perceptual evaluation of voice quality. Increases of jitter and shimmer have been connected to greater phonatory instability.

Horiguchi et al. (1987) reported that the amplitude perturbation measure of the electroglottographic (EGG) signal (shimmer) reflects the irregularity of vocal fold contact during phonation and it could be used as a good parameter of laryngeal pathology. As indicated in Horiguchi et al., perturbations of EGG and acoustic signals in normal subjects and subjects with pathological larynges were measured. Frequency and amplitude perturbation measures (jitter and shimmer respectively) of the voice signal were compared with perceptual ratings and acoustic measures of the voice samples. Their results showed that the shimmer of the EGG signal was highly correlated with auditory-perceptual ratings of hoarseness (0.6968, $p < 0.001$). The amplitude perturbation of the EGG signal showed the highest percentage of correct detection of pathology. Jitter of the EGG signal was highly correlated with that of the acoustic signal in normal subjects but not with the same measure in the patients. Horiguchi et al. concluded that jitter and shimmer in EGG signal, especially shimmer, could be very useful adjuncts to the evaluation of irregularity of vocal fold vibration. According to them, EGG signals facilitate computer analysis because they seem to be simple and do not require pre-processing.

1.7 The motor circuit, the role of dopamine in the motor circuit, thoughts on neural speech control and theories of speech production

It is well established now in neuroanatomy that different circuits or "loops" connect separate cortical regions to the basal ganglia and the thalamic nuclei with specific areas of the frontal lobe. Four such circuits have been identified: skeletomotor (motor), oculomotor, association, and limbic. Martin (1996) uses a practical example (the action of reaching a cup of coffee) to demonstrate the

function of these four circuits of the basal ganglia. The limbic circuit might help in the initial decision to move, the association circuit might help in determining where to direct the grasp of the cup while the motor and oculomotor circuits might help in the planning and guidance of the movement. The origins in each of the circuits are multiple cortical regions with similar general functions. Each circuit projects to different parts of basal ganglia and thalamic nuclei or separate portions of the same nucleus and innervates in separate portions of the frontal lobe. The motor circuit is considered an important circuit for human movement and probably in the movement for speech production. A discussion on the specific neuronal architecture and function of the motor circuit will follow.

1.7.1 Structure and function of the motor circuit

The structure of the motor circuit involves different connections. These connections include structures such as the putamen, the globus pallidus, the substantia nigra, and the thalamus. The putamen have connections to globus pallidus and substantia nigra pars reticulata and from there to thalamus and cortex. The major connections within the motor circuit are:

- The putamen project to specific portions of Globus pallidus external (Gpe), Globus pallidus internal (Gpi), and substantia nigra pars reticulata (SNr).
- GPi and SNr project to specific thalamic nuclei (nucleus ventralis lateralis pars oralis (VLo), nucleus ventralis anterior pars parvocellularis (VApc), and centromedian nucleus (CM).
- VLo and lateral nucleus ventralis anterior pars magnocellularis (VAmc) project to SMA.

- Lateral VApc and VLo project to premotor cortex.
- VLo and CM project to motor cortex.

The major anatomical connections of the motor circuit are in Figure 9 (Duffy, 1995).

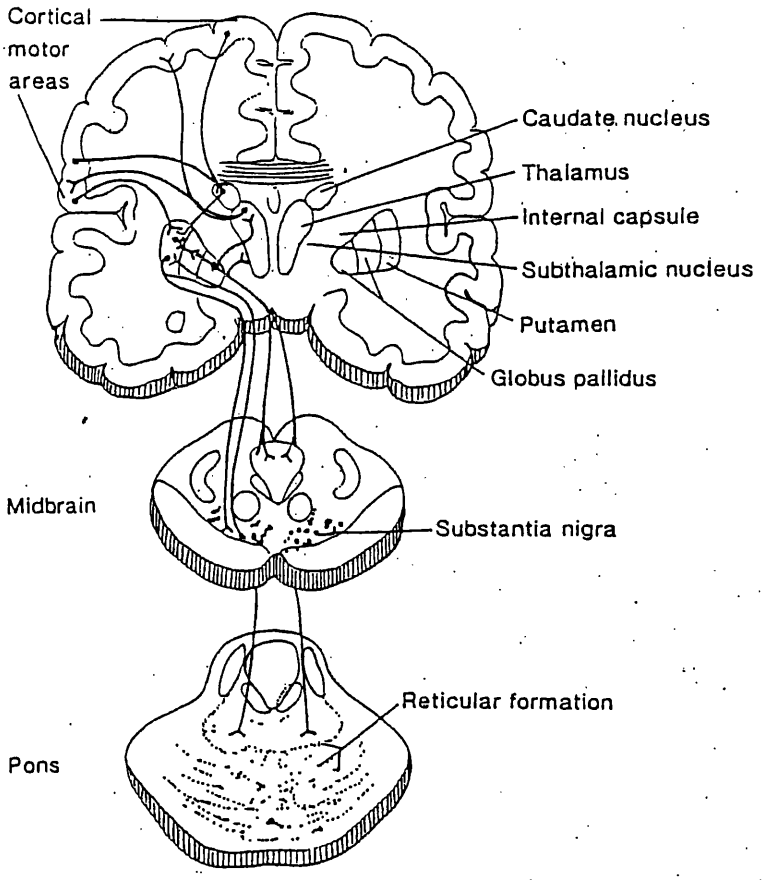


Figure 9. Major anatomical connections of the motor circuit (Duffy, 1995).

Alexander and Crutcher (1990) suggest that there are topographic projections of neurons in the motor circuit. A discussion on their pilot work will follow. In primates, the putamen receive topographic projections from the primary motor cortex (PMC), the arcuate premotor area (APA), and the supplementary motor area (SMA). Other topographic projections to the putamen come from the somatosensory cortex. According to the authors, there is a somatotopic organisation of these projections that consists of a dorsolateral zone

(representation of leg), ventromedial zone (representation of mouth and face) and an in-between them region (representation of the arm). Figure 10 is taken from Alexander and Crutcher and shows the zones and their topographic somatotopic projections to all areas.

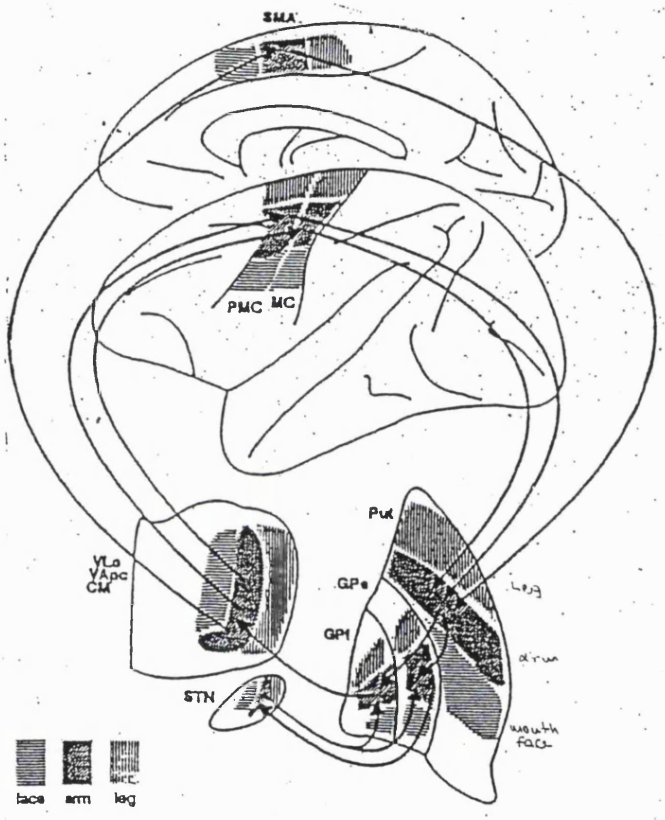


Figure 10. Zones and topographic somatotopic projections between the basal ganglia and different cortical areas.

For example, the arm region of the putamen receives non overlapping projections from the respective arm representations of SMA, PMC, and APA. According to the authors, "there may be separate sub-channels (e.g., SMA- and motor cortex-specific) within each of the somatotopically defined channels (leg, arm, and orofacial) of the motor circuit" (Alexander & Crutcher, 1990, p. 269).

At all stages of the circuit (cortical, striatal, and pallidal) there is a

functional specificity of neurons. Experiments in monkeys showed motor performance of tasks involving a dissociation of the direction of limb movement from the pattern of muscle activity. So, large populations of movement-related neurons act irrespective of the associated pattern of muscle activity but in connection with the direction of limb movement.

The motor circuit may be also involved in the preparation of movement as has been shown in studies with primates in whom premotor cortex, SMA, and motor cortex contain neurons such that their discharge rates change with the presentation of an instructional stimulus. So, individual neurons exhibit movement-related responses or preparatory responses, but not both. During the preparation and execution of limb movements, different aspects of motor processing take place in parallel (in different points within the motor circuit). A sequence of defined levels of motor processing translates the spatial characteristics of the target of movement into an appropriate pattern of muscle activations.

All regions of the circuit (SMA, putamen, and motor cortex) contain different groups of neurons that discharge selectively in relation to variables concerning; the location of the target in space, the direction of limb movement (independent of the muscle pattern), and the force of the movement and/or muscle pattern. The timing of neuronal activity that is related to the various processing levels was found to concur. All of the above suggest the existence of a deeper level of organization within each somatotopic channel of the circuit (leg, arm, orofacial). This organization involves specific sub-channels that encode in parallel but selectively, information about location of the target, limb kinematics and muscle pattern. It is assumed that within these basal ganglia-thalamocortical circuits a functional integration takes place

based upon the temporal coincidence of processing within pathways whose functional segregation is rather strictly maintained. This can be better understood by the simultaneous processing of information relating to coordinated hand and eye movements within the domains of motor and oculomotor circuits.

Alexander and Crutcher (1990) conclude that the functional organization of basal ganglia circuitry reflects the parallel form of neural architecture. Their activity possibly has a unified role "in modulating the operations of the entire frontal lobe, influencing in parallel and by common mechanisms such diverse 'frontal lobe' processes as the maintenance and switching of various behavioral sets (via the prefrontal and limbic circuits) and the planning and execution of limb and eye movements (via the motor and oculomotor circuits)" (p. 270).

1.7.2 The role of dopamine in the motor circuit

Wichmann and DeLong (1993) supported the hypothesis that the symptoms of Parkinson's disease are developed due to abnormalities of the "motor" circuit of the basal ganglia. The loss of nigrostriatal dopamine is responsible for these abnormalities. The input to the motor circuit is parts of putamen while the output is the internal segment of the globus pallidus and the substantia nigra pars reticulata. Dopaminergic projections from the substantia nigra pars compacta modulate the activity in the circuit. Other dopaminergic neurons project to globus pallidus (in external segment mostly and in internal segment to a less degree) and a small number of them terminate in subthalamic nucleus.

Two pathways (direct and indirect) arise from the neurons in putamen and terminate to globus pallidus internal and the substantia nigra pars reticulata

(output of the motor circuit). The direct pathway is considered inhibitory and it projects to the internal segment of the globus pallidus and the substantia nigra pars reticulata, while the indirect pathway is considered excitatory making connections to the external pallidal segment, then to subthalamic nucleus and finally to the internal segment of the globus pallidus and the substantia nigra pars reticulata. Portions of this pathway, primarily to the external pallidal segment and secondary to the subthalamic nucleus, are inhibitory while the portion of the subthalamic nucleus to the output of the circuit is excitatory.

Figure 11 is taken by Wichman and DeLong (1993) and shows the role of dopamine in normal human movement and in Parkinsonism. A detailed explanation follows.

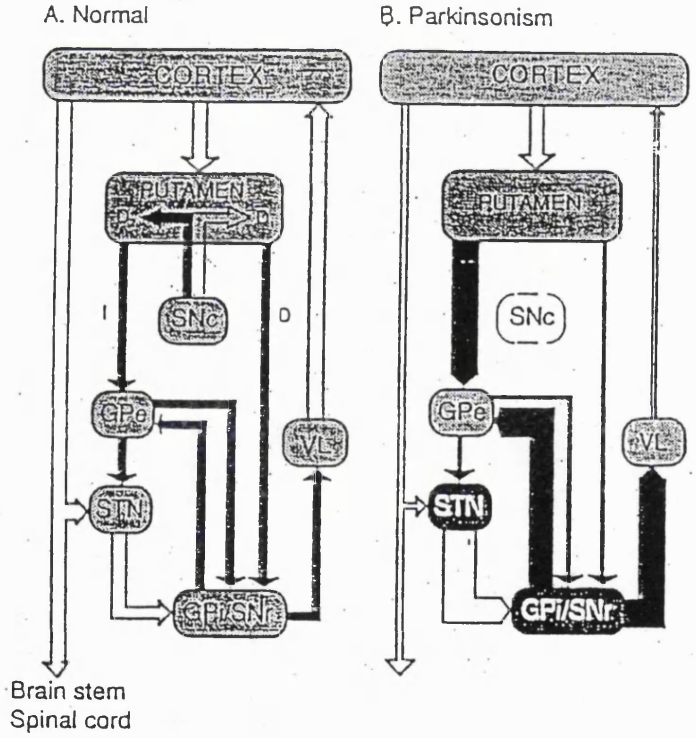
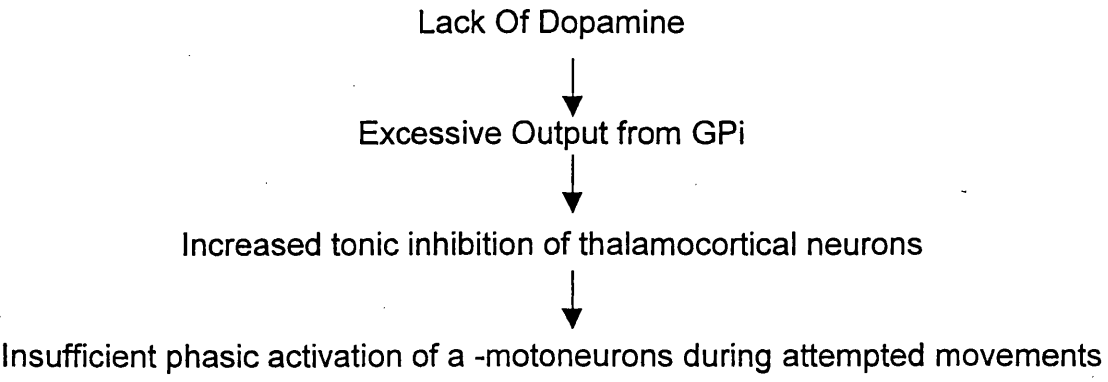


Figure 11. The role of dopamine in the motor circuit and the development of Parkinsonism.

In normal conditions, the role of dopamine is to regulate the activity in the direct pathway and indirect pathway through facilitation of motion in the direct pathway.

It has the opposing effect in the indirect pathway. In other words, dopamine excites the direct pathway and inhibits the indirect pathway. Because these pathways have opposing effects, dopamine tends to facilitate motion. In Parkinson's disease, dopaminergic neurons that are located in the substantia nigra pars compacta and in the ventral tegmental area are destroyed. The lack of dopamine creates the opposite effect (slowness of movement) and it is responsible for the existence of clinical symptomatology.

A detailed explanation of the resulting clinical symptomatology in Parkinson's disease will follow. The excessive output of the GPi is caused by the lack of dopamine. This excessive output results in an increased tonic inhibition of the thalamocortical neurons, which in turn reduce their responsiveness through a reduction of their activity. Finally, this will result in insufficient decline in the frequency of firing of the a-motoneurons during attempted movement (decreased phasic activation).



Marsden (1982, 1994) discusses the general role of the basal ganglia in the normal human movement and in Parkinson's disease. He suggests that the basal ganglia allow the automatic (subconscious) execution (running of a sequence of motor programs to achieve a motor plan) of learned (laid by practice) motor plans. According to him, a motor plan "is the concept of an

action, whose execution requires the sequential operation of a number of simple motor programs" (Marsden, 1982, p. 535). Examples of motor plans according to him are the writing of a signature, the driving of a car, or the riding of a bicycle. The execution of a motor plan is irrespective of the muscles required and involves a series of stages from one point to another "whereas the signal of arrival at each point being the trigger to delivery of the motor program required to shift to the next point in the sequence" (p.535). Marsden states that the basal ganglia are involved in running the sequences of motor programs to complete a motor plan.

1.7.3 Possible relationships of neural architecture and dopamine function to speech production and hypokinetic dysarthria

This section aims to relate the aforementioned reports on neural architecture (structure and function) and the consequences of dopamine dysfunction to speech/voice. Possible relationships about neural architecture, lack of dopamine and hypokinetic dysarthria will involve only assumptions since there is no experimental evidence to prove them.

It is well known that speech production involves a series of movements of the articulators. Speech is considered a complex "product" rather than a simple series of movements involving levels such as semantic, syntactic, morphological and phonological. For reasons of simplicity these levels are not covered in this discussion. Instead, they will be partially covered in the following section that deals with theories of speech production. It is assumed that in the beginning of Parkinson's disease (at least in the sample of Parkinsonian patients that are employed in this study), there are no cognitive problems (Levin et al., 1989;

Levin & Katzen, 1995) that are associated with speech production (Murray, 2000).

Functionally, movement in leg and arm, but more importantly in mouth and face, is regulated through neuronal firing activity in the basal ganglia (Alexander & Crutcher, 1990). The pattern of this activity takes place through a sequence of levels of motor processing that translate the spatial characteristics of the target movement into an appropriate pattern of muscle activation. As has been stated, there is a specificity of function in neuronal groups in the basal ganglia regarding the location of target, the direction of limb movement, and the force of movement and/or muscle pattern. Movement in the mouth and face (and probably the speech movement) should be regulated through the basal ganglia. In speech, this is proven by the existence of hypokinetic dysarthria itself, where there is a dysfunction of basal ganglia. However, the time of the appearance of hypokinetic dysarthria may determine if speech/voice dysfunction is attributed to basal ganglia dysfunction or to other structures involved. Because Parkinson's disease is a progressive neurological disease, it could be possible that hypokinetic dysarthria would be the result of the destruction of other neuroanatomical sites rather than basal ganglia that occur during the disease process. The question to be answered is when dysarthria affects the speech system. If it is affected early in the course of the disease then it is possible that the lack of dopamine creates speech/voice symptomatology in Parkinson's disease as it does in the limb movement.

As stated above, the role of dopamine in normal human movement is to regulate the activity of the basal ganglia pathways. The destruction of dopaminergic neurons creates the clinical symptomatology of idiopathic

Parkinson's disease through an insufficient decline in the frequency of firing of a-motoneurons. This result involves bradykinesia (slowness of movement), rigidity, and tremor. Functionally, the lack of dopamine creates an incoordination of the automatic motor programs to complete a learned motor plan (Marsden 1982, 1989, 1994). Marsden (1982) uses the example of a person who walks through the lobby of a hotel to pay his bill. As he walks he puts his hand in his pocket to take his wallet. The incoordination of the simultaneous activity of the motor programs (walking and putting the hand in the pocket) results in only one of them in time and makes the person to freeze in his position (motor plan was the idea of paying the bill). This incoordination is manifested not only in the simultaneous activity of different motor programs but also in the repetition of the same motor program (Marsden, 1989). Degradation in the repetition of movement as in finger tapping, micrographia, and hypophonia imposes a difficulty in generating long sequences of movement and reveals how the loss of dopamine can influence movement (Marsden, 1989). Under this concept, it would be logical to hypothesize that repetition of volitional movement of the articulators might reveal dysarthric symptomatology in Parkinson's disease.

Along the same lines, Tetrud (1991) assumes that if dopamine is the chief modulator of automatic motor programs then its deficiency might be apparent in complex motor programs such as speech production and handwriting. Anecdotal reports by patients prove this fact (Critchley, 1981; Tetrud, 1991). Also, the majority of studies show that levodopa reverses speech/voice symptomatology (Cahill et al., 1998; Gallena et al., 2001; Leanderson et al., 1971; Mawdsley & Gamsu, 1971; Nakano et al., 1973; Wolfe et al., 1975). However, there are studies that oppose the favourable effect of levodopa (Daniels et al., 1996; Gentil

et al., 1998; Gentil et al., 1999; Poluha et al., 1998). The employment of subjects with variable duration of disease, a moderate disease in neurological symptomatology and different methodological issues may be the cause of these discrepancies. Duration of disease and the existence of motor complications may be confounding factors to the effect of levodopa not only in speech/voice but also in limb movement.

The primary neurological symptom in early Parkinson's disease has been reported to be bradykinesia (Marsden, 1994; Scharre & Mahler, 1994; Tetrud, 1991). In fact, the diagnosis of idiopathic Parkinson's disease involves bradykinesia plus one of tremor or rigidity. It is also clear that rigidity creates the speech and voice symptomatology in hypokinetic dysarthria (Darley et al., 1969a, 1969b; Duffy, 1995; Gentil & Pollak, 1995; Jiang et al., 1999; Kent, 1990; Ludlow & Bassich, 1984). One report however, states that it is bradykinesia that is responsible for hypophonia in Parkinson's disease (Quinn, 1997). It remains to be answered if there is dysarthria in a sample of Parkinsonian subjects in the beginning of the disease where bradykinesia and tremor are the chief neurological symptoms. Tetrud (1991) supports the notion that speech/voice are sensitive measures to detect changes of the disease during its preclinical period, even before the neurological diagnosis. Similar studies in limb movement have found that the time for the upper limb movement as a correlate of bradykinesia was a useful measure to detect motor disturbances in early Parkinson's disease (Watts et al., 1991).

In contrast, Marsden (1994) mentions that speech symptomatology start later in the disease process. It is possible that the discrepancies among these reports lie in the definition of dysarthria. Tetrud (1991) involves voice in

dysarthria while Marsden does not. Medical specialists tend to involve hypophonia (reduction of voice volume) as a distinct entity that does not relate to dysarthria. This is one of the reasons that numerous studies in hypokinetic dysarthria involve different incidence rates (Coates & Bakheit, 1997; Hartelius & Svensson, 1994; Hoehn & Yahr, 1967; Logemann et al., 1978; Mutch et al., 1986; Scott et al., 1985). The primary purpose of this study is to shed a light to these discrepancies and to propose possible explanations. Attempts will be given to explain the findings of this study through a presumed relationship of human movement to speech movement. Under this logic, a detailed discussion of the findings and a proposed mechanism of the speech symptomatology will be further explained in chapter 6.

In conclusion, studies in neuroscience and in neurology probably give important clues to speech pathology because they emphasize the role of basal ganglia in movement. A proposed relation of limb movement to speech movement will be assumed since speech production involves a series of automatic learned motor programs to produce a motor plan (articulate an idea). Even though this presumed relationship has not been considered straightforward by some researchers (Kent, Duffy, Slama, Kent, & Clift, 2001; Murdoch, 2001) their notions are based on evidence of the speech in advanced Parkinson's disease. In contrast, the hypophonia and micrographia that are considered early symptoms of the disease prove this relationship along with studies that found that the simultaneous activity of walking and reciting names was more difficult in Parkinsonian patients than controls in both time and number of steps (Camicioli, Oken, Sexton, Kaye, & Nutt, 1998). A proposed synthesis of the neurophysiological findings with the neurological and speech symptomatology

during the progress of Parkinson's disease is shown below in Figure 12.

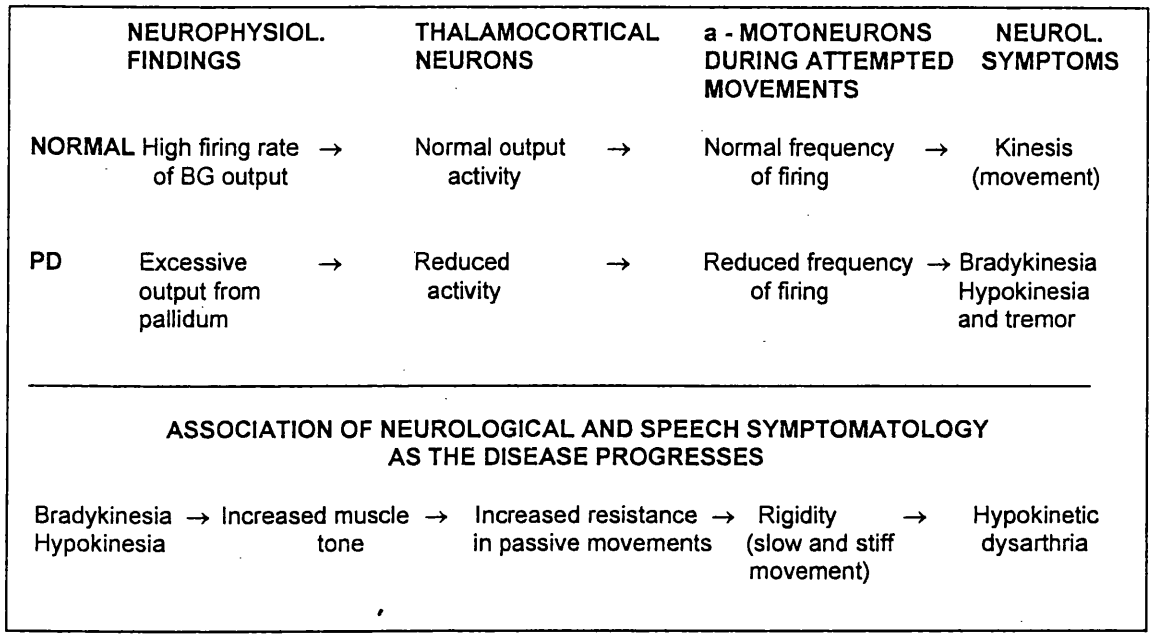


Figure 12. A proposed relationship of neural findings to speech symptomatology in idiopathic Parkinson's disease.

The reduced range of movement can be the result of hypokinesia itself but this did not seem to occur so consistently as bradykinesia in the findings of the present study.

1.7.4 Thoughts on neural speech control

Before giving an introduction to the theories of speech production, it would be appropriate to mention recent thoughts on neural speech control that are based primarily on a review chapter by Kent and Tjaden (1997). An understanding of neural speech control will help to clear inconsistencies or confusion about the role of basal ganglia in the overall complex of speech production. A brief outlook of structures that seem to be considerably important in speech production will follow together with a proposed function between them.

Supplementary motor area (SMA) seems to have a particular role in speech production. The internal generation of movement and the execution of complex sequential movements are listed in the literature, among others, as capabilities of the SMA (Jonas, 1981; Kent, Kent, & Weismer, 2000; Kent & Tjaden, 1997). Kent and Tjaden (1997) hypothesise that if speech could be considered to consist of internally generated movements then SMA would be an active participant in speech control. Jonas (1981) states that the SMA:

- Makes easier the initiation of transmission of ideas (propositional speech)
- Helps in the suppression of non propositional (automatic) speech
- Might be involved in pacing of propositional and non propositional speech
- Can slow speech and cause variability in rate of speech emission.

In general, the control of articulation, phonation and rhythm mechanisms that underlie audible speech involves an intact SMA. Lesions in SMA can cause a different range of speech problems characterised by paroxysmal involuntary phonation/aphonia and articulation.

The concept of neuronal networks that has been discussed in the role of basal ganglia from Wichman and DeLong (1993) seems to influence ideas on cognition, and speech organisation and production. The general logic of a network is the transmission and retransmission of converging outputs from multiple cortical areas to other cortical and subcortical areas through specific functional patterns. In cognition, it is hypothesised that discrete cortical regions might be important for localization in elementary functions while widespread cortical and subcortical networks can be parallel processed for information and complex functions (Bressler, 1995). Bressler (1995) states that "this process may underlie the coordinated transfer of multimodal cortical information which has

been postulated to occur both to the hippocampal region and to the basal ganglia" (p. 299). The same concept is proposed to underlie speech production (Kent & Tjaden, 1997). Paulesu, Frith, and Frackowiak (1993) suggested that SMA, cerebellum, and possibly sensory-motor areas might be involved in a general neuronal network that may be responsible for language planning and execution. Figure 13 below is from Kent and Tjaden (1997) and it describes neural structures and associated neurogenic disorders of speech.

Function	Primary Neural Structures	Disorder
- Intention	Fronto-limbic formations of the forebrain	Mutism
- Linguistic-Symbolic Processing	Cortico-cortical connections	Aphasia, apraxia of speech
- Motor speech Programming or planning	Wernicke's area, Broca's area, premotor cortex, supplementary motor area, inferior parietal lobule, inferior dorsolateral cortex cerebellum, basal nuclei	Apraxia of speech
- Coordination	Basal nuclei, cerebellum, motor cortex	Dysarthria
- Execution	Pyramidal and extrapyramidal motor pathways	Dysarthria

Figure 13. Function, neural structures and associated disorders of speech (Kent & Tjaden, 1997).

Kent and Tjaden (1997) suggest a general framework of cortical and subcortical areas that might be involved in speech production. The inputs of information or neural impulses are the areas motor cortex, SMA, premotor cortex, somatosensory cortex, and superior parietal lobule. After their processing through the basal ganglia these inputs retransmit to premotor fields (SMA, premotor cortex, Broadman's area 44, and cingulate cortex). From premotor fields the inputs are funnelled to motor cortex together with somatosensory inputs from the areas 1, 2, 5. Finally, the output is projected to motoneurons,

premotor cortex, thalamic nuclei, striatum, red nucleus, brain stem and spinal cord (dural and ventral horns). The following diagram that is taken form Kent and Tjaden shows the aforementioned connections.

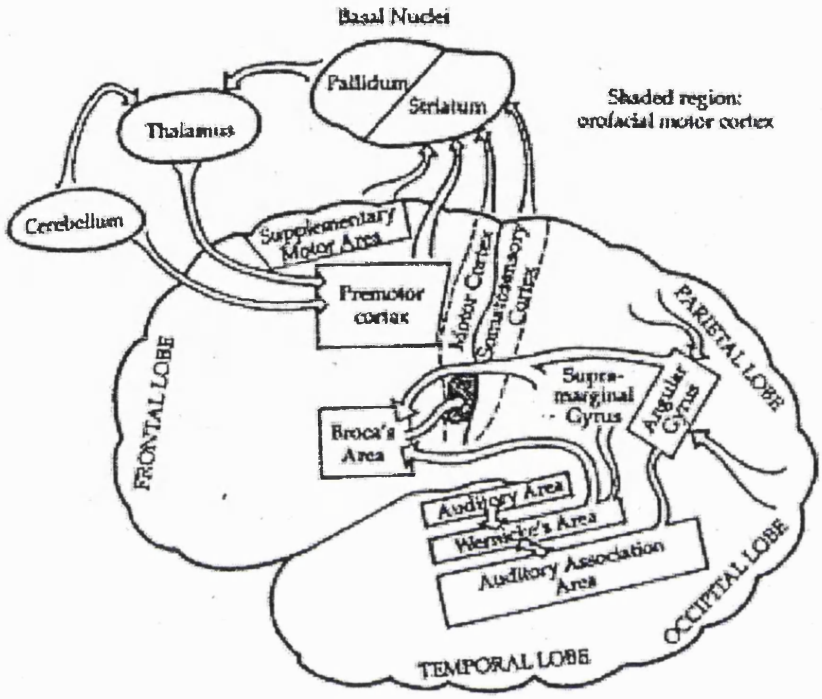


Figure 14. Connections of cortical and subcortical areas that may be involved in speech production (Kent & Tjaden, 1997).

Neuroanatomically, in each structure, there are neural specializations that contribute to its function. Kent et al. (2000) propose that the motor regulation of speech is organised in subcomponents (modules), in which every neuroanatomical site has its own role to speech.

Physiologically, the motor cortex is regarded as the area in which there are representations of muscles. Efferent microzones with each microzone representing individual muscles or muscle synergies are suggested to exist in the motor cortex. Many non contiguous motor cortex areas can give stimuli to a

microzone and so a response will be triggered (Wiesendanger, 1986). Motor cortex is a working funnel for movement information from cortical and subcortical areas and can be considered to work as a keyboard whose keys are specific muscles or muscle synergies. Each muscle or muscle synergy will be represented by more than one key and damage to the whole system (keyboard) will affect the muscle responses, although multiple representation can overcome this result by triggering responses in the context of other movements (Kent & Tjaden, 1997). The motor cortex selects the activation of individual muscles in order to accomplish a motor plan (Kent et al., 2000).

However, it is not clear how all the information that is organised and processed by the motor cortex is produced by the speech production mechanism. Numerous theories of speech production have been proposed to solve this problem. A brief review of some of them will follow.

1.7.5 Theories of speech production

Many theories are trying to answer the question of how speech is organised and produced. These theories struggle to address three major problems: the regulation of serial order of speech, the degrees of freedom, and the context-sensitivity problem (Kent, 1997; Löfqvist, 1997). The regulation of serial order of speech aims to answer what is the minimum unit of speech production and how it is ordered to produce speech. The degrees of freedom are the different movements or the muscle contraction alternatives. The degrees of freedom problem aims to discuss how a system can manage so many different movements (degrees of freedom) to produce a specific motor response. Finally, the context-sensitivity problem is concerned with the variation of sound

production in relation to the context of production.

Stage theories (translation theories) support the notion that speech is produced in a series of stages starting with the translation of a mental representation and ending with an articulatory command (Kent, 1997; Löfqvist, 1997). Information from one stage is passed to the next stage. Stages (from the highest to the lowest level) such as segment specification, feature composition and motor command generation, are involved in these theories. Segment specification is related to language formulation by defining the segments that construct the phonetic message. Feature composition involves the redefinition of the segments as phonetic features. Place and manner of articulation are such features. Finally, motor commands are involved to make features into movements by giving instructions to individual muscles for the duration and length of their contractions.

In stage theories the results of every stage are passed on to following stages in a 'top-down' fashion and this creates a challenging issue "to demonstrate the reality of any one stage in the sequence" (Kent, 1997, p. 408). In other words, every muscle needs to take commands from stages above and this probably creates problems when a series of muscles are needed for an intended sentence to be produced. This phenomenon points to the concept of the degrees of freedom. The control of a motor system that is explained by the translation theories needs an enormous amount of information processing in its higher levels in order to give instructions to specific muscles to carry out the speech production. In contrast to translation theories, research on physiology showed that even complex motor actions are not controlled and coordinated in a purely 'top-down' fashion, but at the lower levels (Wilson & Morton, 1990).

Because this study aims to explain physiological phenomena in speech production (lack of dopamine in the basal ganglia), the top-down fashion of the translation theories does not seem to suit to its purpose.

The action theory of speech production aims to solve the degrees of freedom problem. This theory was thoroughly discussed during the eighties (Fowler, Rubin, Remez, & Turvey, 1980; Kelso, Saltzman, & Tuller, 1986). It states that the vocal tract, including the mandible, the lips, and the larynx, works as a system that is connected by equations of constraints. The components of this system function under this principle. Coordinative structures "marshall the articulators temporarily and flexibly into functional groupings of joints and muscles that can accomplish particular goals in speech" (Kelso et al., 1986). Löfqvist (1997) adds that the coordinative structures are coherent patterns of muscle activity that are set spatially (same set of muscles could be activated), temporally (synchronous movement and stable order) and connected with scaled relationships.

The notion of coordinative structures resembles ideas from physiology that the brain is organised into efferent microzones innervating into muscle synergies (Wiesendanger, 1986). The muscle synergies or the idea of a motor area that works as a "keyboard" points to the concept of the coordination of muscles. A group of muscles or a group of muscle collectives functions as a coordinative structure (muscle synergy) in which "indices[2] of the individual muscles or individual muscle collectives appear to covary in terms of a relatively fixed relationship that is indifferent to overall magnitude changes in the indices" (Kent, 1997, p. 394). Wilson and Morton (1990) state that "individual muscles or

[2] According to Kent (1997), the index of a muscle is defined as the level of innervation or the time relative to the group of muscles behaviour at which a muscle is innervated.

muscle groups will be constrained to act in a coordinate fashion by the output of subcortical systems for pattern generation" (p. 346).

Figure 15 is taken from Wilson and Morton (1990) and shows the organization of coordinative structures. In this figure "m" represents a muscle and "CS" a coordinative structure. In this 'nested' organisation the coordinative structures at the lower level form elements at the higher level tied up with equations of constraint. In this organisation lies the basic difference between the action theory and the translation theories (Wilson & Morton, 1990).

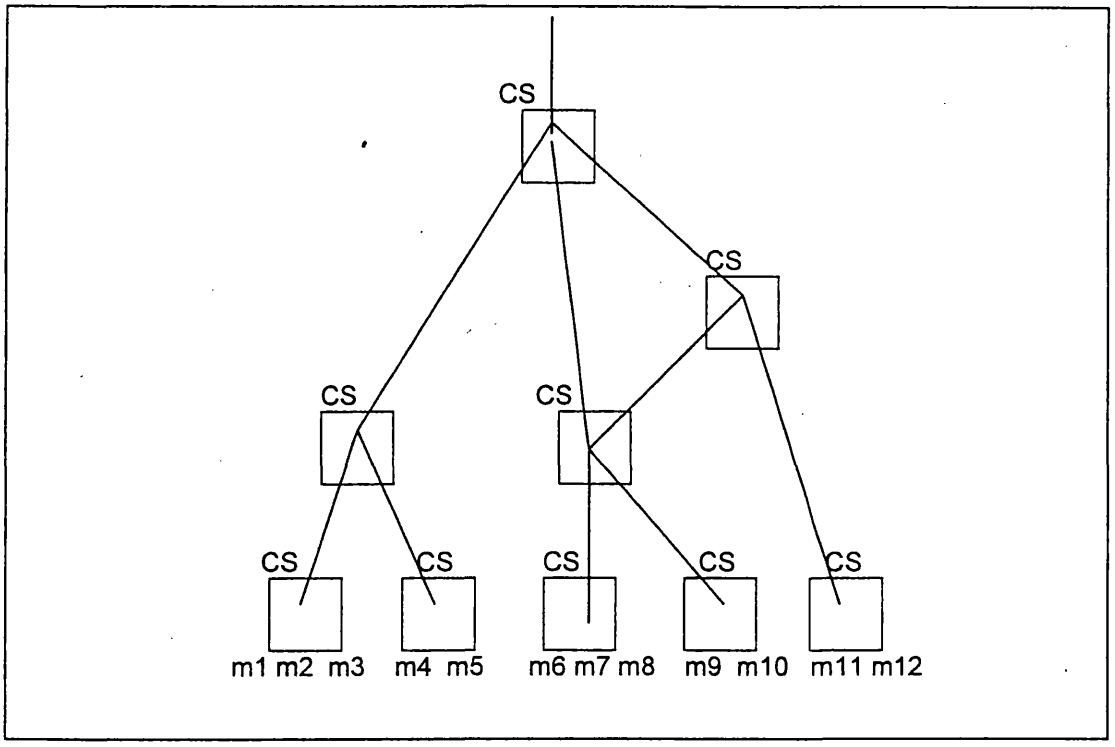


Figure 15. Nested coordinative structures (taken from Wilson & Morton, 1990).

Neurophysiologically, Grillner (1982) supports the notion that locomotion is attained by a system with a 'nested' structure as it is shown above. This structure is responsible for coordinating all necessary muscles and muscle groups to achieve a walking pattern. One nesting (the most basic) is called 'unit central generator' or 'central pattern generator' (CPG) and it is responsible for

controlling the rhythmic activity among the members of its respective muscle groups. CPGs provide the necessary rhythm for life functions such as respiration, locomotion, and mastication (Chandler & Goldberg, 1984).

Speech is viewed as a complex function that is based and developed on functions such as mastication. Gracco (1992) hypothesises that a central rhythm generator provides the framework on which the speech patterns (phonemes) sequence. It is generally hypothesised that CPGs are present in the brainstem and so, it is there that the centre for sequential speech production exists. The unt central generators do not have the need to be controlled every single time from a higher level. Instead, they have the neural mechanisms for independent bursting output and independent control of muscle phasing or coordination of muscle action. A central pattern generator network activates a number of unit generators that are responsible for the coordinated activity of muscle groups and joints throughout the limb. The function of the motor-control network involves a scaling of constraint in which the number of specific elements in the system that must be controlled to achieve patterned motor action at output is reduced at the nested levels.

Equations of constraint govern the functioning of the coordinative structures. In particular, the coordination of muscles depends on the specification of the equations of constraints. An equation of constraint is the base at which individual muscles will work. Wilson and Morton (1990) provided an example of an equation of constraint.

GOAL $\leftarrow$(0.2 of muscle 1) $\times$ (0.7 of muscle 2) $\times$ ...(n of muscle j)

In this example, the goal is a particular position of the tongue following the action of several extrinsic tongue muscles. A change in one parameter of the coordinative structure for the achievement of the goal will create reciprocal changes in the activity of the other elements of the system, and so, the goal will remain constant.

If one of the variables in the equation of constraint is fixed, the other variables will adjust to meet the fixed value of the equation. If for example a bite block is inserted in the mouth during the production of a vowel, the muscle synergy will be adjusted to take values that will attempt to preserve the vowel quality (if it is attainable). If this happens the effect of the bite block will be negligible. For example, the closure of the lips can be attained in different ways:

- The contraction of the muscles of the upper and lower lips if the jaw is immobile
- The cooperation of the jaw if it is free with the lip muscles or
- The movement of the jaw to hold the lips if the lip muscles are inactive (Kent, 1997).

Another theory that attracted much attention after the late eighties is the theory of spreading activation of retrieval in sentence production (Dell, 1986). This theory is a part of a general framework of theories, the so-called connectionist theories. An alternative name for these theories is neural networks because of their assumptions on the organisation of information to resemble neural circuits (Kent, 1997).

The spreading activation theory of retrieval in sentence production attracts more attention because of its modelling application of speech errors in some neurological disorders including the apraxia of speech (Kent, 1997). A

description of this theory will follow based primarily on the work by Dell (1986) together with possible inferences for speech errors in hypokinetic dysarthria.

The roots of this theory are in spreading activation models which hypothesise the existence of a network of linguistic units and rules. In Dell's theory, this network contains nodes for linguistic units (concepts, words, morphemes, phonemes, and phonemic features) that are connected to each other without an order and together represent the lexicon. Dell's theory is primarily based on three general assumptions about the information:

- It is stored in the lexicon
- It is represented as generative rules at different levels and
- There are additional rules that connect the lexicon and the different levels (insertion rules).

A principal assumption of this theory is the existence of different levels and the construction of each planned utterance at various times in each level. Dell (1986) describes five levels: semantic, syntactic, morphological, phonological, and motor. The ordering of the differential representation in different levels of this theory presents similarities to the translation theories. One of the basic differences between them seems to be the existence of nodes in each level of representation.

Each level is the final product of rules (generative rules) operating in it. These rules define or codify the language specific acceptable combinations of items at each level. The syntactic rules represent syntactic categories (noun, verb, etc.), the morphological rules represent morphological categories (stem, prefix, suffix, etc.), and the phonological rules represent phonological categories (initial stop, vowel, etc.). This codification is created by frames and categorised

slots. Finally, the selection of words at the syntactic level, the selection of morphemes at the morphological level, and the selection of phonemes at the phonological level is attained by insertion rules that operate to connect the lexicon and the generative rules. A description of how each representation is built will follow. Figure 16 represents the frames and slots in each level using an example sentence by Dell (1986).

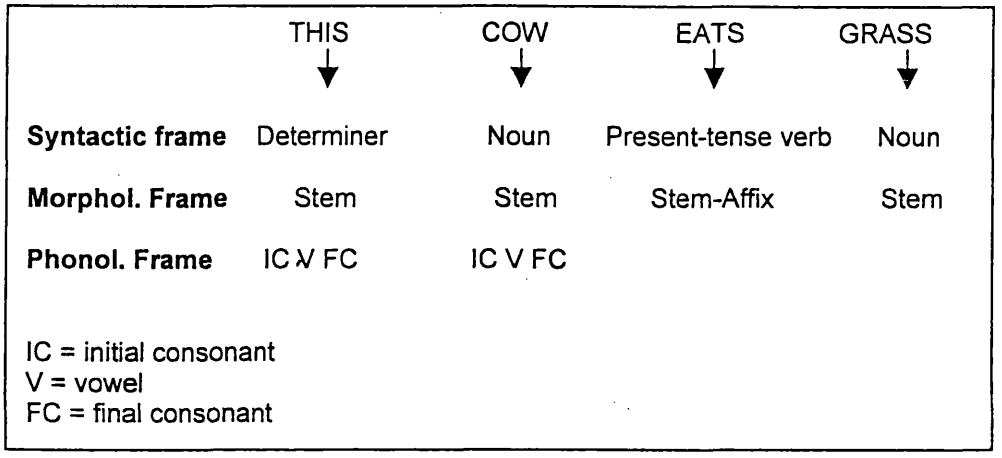


Figure 16. Frames and slots at each rule system operating at each level of representation.

Each representation at each level consists of an ordered set of items that exists in the lexicon. Dell (1986) describes it as "a collection of ordered tags that are attached to nodes in the lexical network, dictating the contents of the representation and their order" (p. 287). The construction of a higher representation precedes the construction of a lower representation. A spreading-activation mechanism helps in the processing of activation of nodes that may be used for a lower representation while the higher representation already has its own tagged nodes. So, a frame for the lower representation is built by the generative rules and the insertion rules fill in its slots through spreading activation. Spreading activation is considered the major mechanism of prediction

of speech errors in Dell's theory. An item selection for a slot, results in the receipt of a tag. The rate of processing depends on the mechanism of the level and the level above it.

The mechanism of spreading activation is based on processes such as:

- Spreading (it is defined as the process by which a node with an activation level greater than zero sends a proportion of its activation level to all nodes)
- Summation (it is defined as the adding to the activation level of a node of the activation that this node receives from other nodes) and
- Decay (it is defined as the natural decay of activation and it is determined by the time of its activation).

According to Dell, spreading occurs when a node has an activation level above zero, and decay occurs when a node has an activation level toward zero. The theory speculates that the construction of the lower representation begins after the node of the higher representation is tagged. An important hypothesis of the theory is that the connections between the nodes are two sided (from node A to B and from node B to A) in order for the message to deliver positive feedback from the later stages to the earlier stages. Excitatory rather than inhibitory processes are involved in the theory.

According to Dell (1986), articulatory errors generally happen from the function of spreading activation and the construction of multiple representations. As it is logically expected the process of articulatory errors involves higher activation of incorrect items instead of correct items. Speech errors can be explained by this theory through factors such as output biases (tendency of speech errors to create meaningful combinations of units), similarity effects

(when interacting items or words in an error tend to be similar), speech rate effects (when there is not enough time for the retrieval of the correct items), and distance effects (when misordered sounds tend to move to adjacent content words that are in the same phrase).

In general, none of the theories that mentioned in this section have a direct association with hypokinetic dysarthria. This is because the articulatory errors in hypokinetic dysarthria are predicted to be more as distortions of the phoneme target rather than substitutions or deletions. More specifically, the errors appear to reflect an inadequate tongue elevation to achieve complete closure on some phoneme categories (stops and affricates) and less constriction (open aperture) in fricatives (Logemann & Fisher, 1981). A general theory of speech motor control needs to include internal models of the articulators, rhythm-based sensory-motor integration and specification of articulatory dynamics within a motor program (Kent et al., 2000). Up to now, no such a theory has explained hypokinetic dysarthria of Parkinson's disease.

Translation theories have the disadvantage of the degrees of freedom problem and serial ordering to explain physiological phenomena. The action theory addresses the degrees of freedom problem but still has not address yet problems in dysarthria. The spreading-activation of sentence retrieval theory has been applied to apraxia and conduction aphasia and its modelling performed fairly well for errors in normal speech (Kent, 1990). However, no apparent connection of this theory with dysarthric errors has been modelled.

However, Kent and his colleagues hypothesise the existence of sensory trajectories or templates that include patterns of auditory, proprioceptive and tactile information and precede the motor commands for speech production

(Kent, 1990; Kent et al., 2000). These trajectories are assumed to be the next stage of the phonological level in Dell's theory and help in the motor realisation for speech. Kent assigns a sensorimotor role to the basal ganglia and hypothesises that damage to the sensory trajectories due to impairments in afference, together with the motor role of the basal ganglia, are responsible for hypokinetic dysarthria. This hypothesis comes in contrast to the ideas by Marsden (1982, 1994) about the unitary motor function of the basal ganglia in human movement. In any case, it is doubtful (even though not improbable) that impaired sensory information occurs in the beginning of Parkinson's disease (as the present study examines).

1.8 Conclusion and research questions

The present study aims to investigate the existence of hypokinetic dysarthria in the beginning of Parkinson's disease. The perceptual features that were reported to be more prominent in hypokinetic dysarthria were monopitch and monoloudness (Darley et al., 1969a, 1975). The advent of new techniques (electrolaryngography), might be able to measure quantitatively aspects of voice such as fundamental frequency and intensity that correlate to the perceptual impressions (monopitch, monoloudness) in various tasks including sustained phonation, reading, and conversation. Dysarthria and intelligibility as parts of a motor speech examination are also addressed and possible inferences about movements (speech and non speech) of the articulators are emphasised.

More specifically, the experimental questions that are addressed here involve:

1) What is the incidence of hypokinetic dysarthria in patients recently diagnosed

with idiopathic Parkinson's disease as defined by the Frenchay Dysarthria assessment and intelligibility?

2) What is the effect of Parkinson's disease on speech/voice parameters in patients early diagnosed with idiopathic Parkinson's disease and compared to a control group matched by age and gender?

3) What is the effect of medication on speech/voice parameters of the dysarthric Parkinsonian group 3-3.5 months after the neurological and speech diagnosis?

CHAPTER 2. BACKGROUND TO THE METHODOLOGY

The purpose of this chapter is to analyse and further justify the decisions that were taken during this study. This chapter aims to explain the choice of the research design, the use of the particular instruments, and the control of extraneous factors (vision, hearing, and posture). It was developed using the logic of a critical examination of the literature and the procedures of research study, which are outlined in chapter three. In order to facilitate its reading the sections in this chapter are organised in much the same way as the order of the motor speech assessment procedure.

In contrast to chapter 3, which describes the current study, this chapter analyses and discusses in detail the methodological advantages and disadvantages.

2.1 Matching criteria in the experimental/control group and the validity of the research design in this study

This section aims to explain the reasons for the inclusion of a normal control group to the experimental group, the criteria of matching and other factors that may affect the internal and external validity of the study.

In general, Parkinson's disease is considered a geriatric disease. In later developmental stages of life, general health problems (e.g., diabetes) and specific health problems (hearing, vision) may arise. Among the geriatric population, there is a greater variability in the appearance of these health problems and a reported variability in speech performance when older subjects were compared to young

subjects (Liss, Weismer, & Rosenbek, 1990; Weismer, 1984a; Weismer & Martin, 1992). The inclusion of a normal geriatric group to be compared to the Parkinsonian group in speech/aspects of voice aims to counterbalance this variability.

Pair matching is a technique that is used to minimise the differences between populations. It generally provides more control than matching around the mean (Schiavetti & Metz, 1997). Pair matching is considered a method that increases the sensitivity of the study to small effects of the independent variable to dependent variable (Schiavetti & Metz, 1997). In the present study, pair matching was selected to decrease the variability that may contribute to the measurement of speech/aspects of voice between the two samples (Parkinsonian and control). Differences such as age, gender, and education were considered in the pair matching.

The criteria of matching were as follows:

- In age: a difference of less than 3 years between the pair members
- In gender: no sex differences between the pair members
- In education: fluent reading as defined by at least 4 years of education at elementary level. As such, emphasis was given to finding pairs with no difference between the pair members in terms of education: elementary education (4-6 years), high school (12 years), and university level (more than 12 years).

Age and gender matching criteria are used in most of the studies that compare an experimental and a control group. Education was added as a criterion in the present study because similar reading abilities between the subjects (Parkinsonians and

controls) were considered essential to evaluate voice variables through a reading task.

In theory, mood disorders may also influence and be influenced by speech but more specifically voice (Aronson, 1990; Darby, Simmons, & Berger, 1984). The voice changes are prominent in affective disorders (psychiatric syndromes) exhibiting narrow pitch range, infrequent pitch changes, reduced stress or emphasis patterns and harsh voice quality (Aronson, 1990). Distinctive speech patterns that were improved after pharmacological treatment were found in bipolar and unipolar disorders (Darby et al., 1984). However, the association between speech disturbance and scores on the Hamilton depression scale was found to be non significant (Darby et al., 1984).

In Parkinson's disease (PD) the prevalence of depression is about 40% as shown in review studies (Cummings, 1992; Poewe & Luginger, 1999). Depression has been found to be unrelated to the duration/response to levodopa therapy (Dooneief et al., 1992) and the disease duration (Cummings, 1992; Rojo et al., 2003). Poewe and Luginger (1999) discuss if depression is an early or late feature of PD. They report that there are two peaks in its occurrence. The beginning of PD and its late advancement affect depressive episodes. Also, advanced Hoehn and Yahr staging was found to be associated with depression (Rojo et al., 2003) while the opposite was reported in another study (Cummings, 1992). Finally, depression has also been associated with low scoring in Mini Mental State Examination (MMSE) (Rojo et al., 2003).

Diagnostic difficulties may affect the results in the prevalence of depression (McDonald, Richard, & DeLong, 2003). A deficit in facial expression of patients with Parkinson's disease due to motor abnormalities (akinesia or bradykinesia) may be

perceived as depression in the absence of actual depression (Poewe & Luginger, 1999).

Although speech/voice have been associated with depression (Aronson, 1990; Darby et al., 1984), only one fairly recent study has been found to examine their association with the depression of Parkinson's disease (Sapir et al., 2001). This study aimed to associate perceptual ratings of speech/voice with other variables including depression in 42 Parkinsonian patients. Only 6 out of these patients had a disease duration of 1 year (average disease duration = 7.1 years) and only one was in stage 1 of the Hoehn and Yahr scale (average Hoehn and Yahr stage = 2.6). The results of this study showed that the prevalence and number of voice and speech abnormalities were unrelated to depression scores.

In summary, further research is needed to associate speech/voice abnormalities to depression in Parkinson's disease. The results by Sapir et al. (2001) show that depression in PD does not affect phonation in subjects with moderate stages of Hoehn and Yahr scale and a mean of 7 years of disease duration. However, it seems reasonable to hypothesise that in the absence of more studies to examine this area, the assessment of depression needs to be included in studies examining speech/voice in neurological diseases. It is a limitation of the present study that no assessment of depression took place, although the possibility of its occurrence seems to be low according to the aforementioned studies.

2.1.1 Internal and external validity between groups

The main purpose of the between subjects design is to eliminate influences of extraneous variables that can threaten the internal and external validity of the results. In every research study, the internal validity is dependent on the degree to

which the between groups design has accomplished what it set out to accomplish. The external validity asks about the degree to which the results can be generalised.

In internal validity, test-practice effects, instrumentation, differential selection of subjects, and the Hawthorne effect may crucially affect the differences between the Parkinsonian and the control group. These factors will be explained in detail below.

In the current study, familiarity with the assessment items (test-practice effects) between the two groups was not considered a factor that could influence subjects' performance because each assessment session involved different assessment tools. The first session involved the completion of the history form and the administration of the Mini Mental State Examination (MMSE). The second session involved the administration of the Frenchay Dysarthria Assessment (FDA) and intelligibility testing, and the third session involved the recording using electrolaryngography (ELG).

The administration of the tests took place in the same order for each subject to avoid variability in measurement (instrumentation). With regard to the testing, the Frenchay Dysarthria Assessment (Enderby, 1983) and the Mini Mental State Examination (Folstein, Folstein, & McHugh, 1975) are standardised tests. The intelligibility testing involved a list of words based on minimal pairs for both the experimental and the control groups. The subject's reliability in intelligibility estimation was not an issue in this study because all subjects scored above 90%. With regard to instrumentation, the electrolaryngograph was a calibrated machine newly purchased specifically for this study. With regard to the conditions of the electrolaryngographic recordings, the placement of the equipment and the location

of the recordings were the same for all subjects. Efforts were made to ensure that the control and experimental subjects were recorded at the same time of the day.

As stated above, the experimental subjects were matched in pairs to the controls so no differential selection threatened the internal validity. Changes in behaviour because of knowledge of experimental participation might have been a threat to the present study (Hawthorne effect). These changes might have occurred because the psychological state of the experimental subjects was not appropriate in the first session for a person who has just been informed that he/she has Parkinson's disease. For this reason, a general conversation was carried out between the subject and the experimenter before the assessment, especially during the first meeting, to acclimatise them to the research situation.

External validity is a problem for all comparative research, especially when human beings are measured in performance. Reactive arrangements are factors that can be a threat to external validity. Reactive arrangements on the part of the subjects involve the degree to which the setting of the research might react with the classification variable (Parkinson's disease) in determining the subjects' performance on the criterion variable (speech/aspects of voice). For example, reactive arrangements in the current study involved the use of a quiet place for the recordings. Careful consideration was taken to exclude other influences such as telephone ringing. The electrolaryngographic measurements were all taken in the same place (the examiner's office), at a specific time and with each subject in the identical position. Even though no consideration was given to controlling the level of

noise in the room (use of a sound level meter)[3] the necessary precautions were taken (closed door and windows and the absence of third parties during the recording).

2.1.2 Internal validity within group (medication state)

After discussing the factors that could affect the internal and external validity in the between - groups design, attention must also be given to factors in the within -group design (the effect of medication). In general, the within subjects research design is considered a more powerful design than the between - groups design because the experimental subjects act as their own control group by participating before and after medication. However, sequencing effects (order effect and carry-over effect) may affect the performance of subjects.

The order effect involves an improvement or a decrease in the performance of each subject either through learning the task by participating in it or because of fatigue. The fatigue in the present study was controlled by splitting the examination into 3 consecutive sessions of a short duration (one for every day). The provision of different tasks of short duration in every session controlled the learning effect. The carry-over effect involves the influence of one task on subject's performance on the subsequent task. In this study, this involved the period of time that elapsed between sessions. In the pre-medication state, the first session that involved the completion of the history form took place in the hospital and did not last more than 40 minutes. The second session took place in the home of the subject and lasted no more than

[3] The sound level meter is calibrated in dBs with a reference level to provide accurate measurements of intensity. In the absence of a sound level meter, relative intensity of the voice signal was measured. The electrolaryngographic fundamental frequency measures are not affected by extreme noise.

an hour. Finally, the third session took place in the researcher's office and did not last more than 30 minutes. Each session involved different aspects of performance and so the subject's interest was assured. Because this was the first time that such a study was being carried out in Greece, the subjects reacted positively. In the post-medication state, the sessions took place after 3-3.5 months and the subjects could not remember the content of the previous test. In conclusion, fatigue, order and carry-over effects did not seem to affect the external validity of this study.

2.2 Hoehn and Yahr neurological scale

The Hoehn and Yahr Functional Rating Scale (Hoehn & Yahr, 1967) has been used in the majority of neurological studies to indicate level of severity in Parkinson's disease. A short description of this scale will follow.

Hoehn and Yahr (1967) stated that their scale has two basic advantages: it is practical and it allows for reproducible assessments by independent examiners. This scale ranges symptomatology from mild to severe disability. It is divided into 5 stages (I - V). In each stage the symptomatology progressively worsens until the final stages. For example, stage IV involves the existence of rigidity, akinesis, poor standing balance, and poor fine motor coordination with severely disabling results for the patient (affecting ambulation at home and the need for assistance in self-care skills). In contrast, stage III involves a festinating gait but the attendant's assistance is not as frequently required. In this stage the patient's disability is manifested when he/she starts, turns, stops, and steps backward. According to the authors, a longer duration of the disease is associated with the later stages. A description of the scale cited by Yorkston et al. (1995) is given in Appendix B. In the present study, all experimental subjects were in stage I of Hoehn and Yahr scale.

According to Hoehn and Yahr, the symptoms of the disease in stage I involve mild resting tremor, rigidity, bradykinesia, dysarthria, trunk tilt, fine motor incoordination, and facial immobility. These symptoms are unilateral and just noticeable (not disabling).

2.3 Exclusion criteria in this study. Factors other than the experimental variables controlled for in the study

The exclusion criteria involved developmental problems of speech and language, visual problems, hearing problems, reading problems, and cognitive problems. The existence of each one of these extraneous factors could add doubtful matching between the subjects and could lead to arbitrary confounded results. A discussion for the control of these factors together with factors that could influence the quality of the recordings will follow.

2.3.1 History of speech and language problems, vision, hearing and reading skills

A previous history of speech and language problems, inadequate vision, impaired hearing, and inadequate reading skills were determined as factors that needed to be controlled in this study. Also, present history of laryngeal pathology and levodopa medication before the neurological diagnosis, were also taken into account. It is a common practice in almost all studies of dysarthria to control the aforementioned factors. All these factors were assessed informally through the administration of the history form and through a conversation with the subjects and relatives.

Questions involved the existence of any speech and language problems during childhood and whether these problems still interfered with the subjects' lives. Other questions explored the existence of present hearing and visual problems that could affect performance in the tasks of the study. Finally, the reading ability of the subjects was assessed through questions during the interview and the assessment of cognitive status. Illiterate subjects were excluded from the study. The task that was predicted to interfere most with all the aforementioned factors was the reading of a Greek passage. This passage is complex because it involves most of the consonants and consonant clusters in almost all positions in the Greek language. Two subjects were found to exhibit a present history of laryngeal pathology (PX9) and a past history of levodopa medication (PM7). For this reason, PX9 was not compared with her matched pair control (in the between - groups design) but she was used in the effect of medication to see if levodopa medication had a differential effect on her speech/voice. PM7 stopped levodopa medication 6 months before the speech/voice assessment. Because this period of time is felt to be insufficient to interfere with his speech/voice, this subject was included in the sample.

2.3.2 Cognitive status-The Mini Mental State Examination (MMSE)

The Mini Mental State Examination (MMSE) is a quick test to assess cognitive function and it is especially useful in the recognition of dementia in patients (see Appendix C for the English and Greek copies of the test). In its original form (Folstein et al., 1975), it was proposed as "a quantified assessment of cognitive status of demonstrable reliability and validity" (p. 195). MMSE has been used around the world and has been proved to be robust to assess cognitive ability. Furthermore, it has been used as a core cognitive measure in a number of major

epidemiological studies and instruments including the Medical Research Council Cognitive Function and Ageing Study in the United Kingdom (Shulman & Feinstein, 2003). Its construct validity in longitudinal studies of dementia shows a decline of scores (2-5 points) per year in subjects.

In most of the studies, the MMSE cut-off value that indicates cognitive disturbance is 23 or 24 out of 30 (30 being the maximum score). Anthony, LeResche, Niaz, Von Korff, and Folstein (1982) found that the sensitivity (probability of correctly identifying any case) and specificity (probability of correctly identifying any non case) percentages for the MMSE scores (from 0-23) were 87% and 82% respectively. According to them, the test was highly specific in detecting delirium and dementia in subjects less than 60 years old with more than eighth grade level of education. However, some limitations of the MMSE have been reported.

The MMSE's total score proved not to be a strong predictor of cognitive status in 251 Swedish adults over 75 years (Hill & Bäckman, 1995) and in subjects over 85 years, subjects from manual social class and subjects with some visual impairment in the United Kingdom (Jagger, Clarke, Anderson, & Battcock, 1992). However, when taken separately, the questions that test memory, spatial skill, and the ability to follow commands proved to be good predictors of cognitive ability (Hill & Bäckman, 1995).

Grigoletto, Zappalà, Anderson, and Lebowitz (1999) aimed to find norms for the MMSE in 908 healthy adults aged 20-79 years. The authors found that the norms proved to be higher in the population with higher education. Elderly women with a low educational background exhibited difficulties during the administration of MMSE. Similar to the other studies, this study proved that low educational

background, age, and gender were important factors for lower performance in the MMSE.

The current study used the Mini Mental State Examination (MMSE) to assess cognitive ability. However, the Parkinsonian subjects expected to be close to normal in MMSE scoring, being at the beginning of the disease. Impairments in executive function in Parkinson's disease coincide with stages 2 and 3 in Hoehn and Yahr scale and with the appearance of bilateral symptomatology (Kanazawa et al., 2001). So, the use of MMSE was a further factor of insurance of no cognitive impairment at the beginning of Parkinson's disease, even though MMSE is not considered a detailed test for cognitive assessment (see a recent discussion by Shulman and Feinstein, 2003). The score below 24 was used as a cut-off score for cognitive status. Only one subject was excluded from the study (KM12). This subject had a low educational background, and as expected she exhibited a lower performance. Moreover, the appearance of other factors in this subject (hearing problems) made any further testing unnecessary. One subject (SB5) was above 75 years of age but he was from an upper social class and with a higher education.

In conclusion, the existence of dementia/cognitive problems in the present study was examined in two ways. First, during the administration of the history form that took place by checking the accuracy of the demographic and other information provided by the subject together with a relative who accompanied the subject. Second, through the administration of the MMSE and its scoring. Therefore, none of the prohibiting factors of cognitive score that mentioned in the aforementioned studies, seemed to affect the present study.

2.3.3 Posture, microphone, setting, and location during recording

During recording, extraneous variables such as posture, location, and microphone distance were stable. In this study, the control of these variables was similar to other studies, especially acoustic studies that used the same standard procedures. With regard to posture and setting, it was necessary for the subjects to sit on a chair either in their homes (second meeting) or in the office of the experimenter (third meeting). During the administration of the dysarthria assessment and the consequent intelligibility scoring, the recording took place at the subject's house in a quiet room and it was attempted to ensure that noises such as the telephone would not disturb its quality. It was imperative to the current study that the electrolaryngographic recordings would be carried out in the same location (the office of the examiner) and that all subjects would be seated in the same position.

With regard to microphone distance, different procedures took place in the assessment of the intelligibility score and in the electrolaryngographic measurement. In the intelligibility assessment it was necessary for the subject to read one word on each card that was presented by the examiner while his/her speech was tape-recorded through a stereo microphone located on a boom stand on the table. The experimenter asked the subjects to keep a constant distance from the microphone (about 15-20 cm). During the electrolaryngographic recordings, a standard distance from the microphone to the mouth of the subject was maintained at all times. The microphone was attached to velcro about 12 cm from the electrodes (on the neck of the subject). The subjects were asked to move their heads as little as possible during recording in order to keep the microphone

distance stable and to ensure the best quality of electrolaryngographic measurement.

2.3.4 Time of assessment

In general, the time of the day in neurological studies is considered an essential factor for the success of the study, because of the influence of the disease on the physical state of the patient during the day. In many studies the subjects' relatives have been reported to observe that "his/her speech worsens in the evening and at night".

In the present study, the above observations were taken into consideration even though the experimental subjects were at the early beginning of the disease and it was predicted that they would not exhibit such fatigue problems. Most of the recordings took place in the morning (approximately 10:00-11:00 am). However, there were cases when the experimental subjects were unable to come to the examiner's office due to work commitments. In such cases, the recordings took place in the early evening (6:00-7:00 p.m.). In order to control for such changes of time within the experimental and control groups, efforts were made to record control subjects' speech/voice at almost the same time as their matched experimental subjects.

2.4 Motor speech examination and the development of the history form

An analytical motor speech examination should include the following:

- A completion of a history form (which the examiner will go through with the patient and/or relatives during the interview)

- An examination of the speech mechanism during non speech activities
- An assessment of speech intelligibility
- An acoustic analysis including vowel prolongations, Alternating Motion Rates (AMRs), reading of a passage, and conversation.

The development of the history form will be discussed in this section while the examination of the speech mechanism during non speech activities, the assessment of speech intelligibility and the acoustic analysis of speech are explained in the subsequent sections.

The development of the history form requires detailed knowledge of specific parameters that may have crucial importance to the motor speech examination. Such parameters involve detailed biographical data, facts about the onset and course of the speech symptomatology and other relevant data. The history form is based on the patient's and/or family's perceptions of the speech problem.

Duffy (1995) suggested a list of factors that a history form should include. The current study used these suggestions in the development of the history form. These factors were organised in the form of groups of questions to the subjects. The first group included biographical information about age, education, occupation, marital and family status, developmental speech/language problems, and visual/hearing problems if they existed. The date of diagnosis as well as the duration for the completion of the history form was also included.

The second group of questions included information about the onset and course of the speech deficit and the patient's perception of it. Questions that were considered useful included:

" Do you have any difficulty with your speech? If not, has someone else commented on a change or problem with your speech?" (Duffy, 1995, p. 68).

"Has the speech problem changed over time? Is it better, worse, stable, better-then-stable, fluctuating?" (Duffy, 1995, p. 68).

This group of data also included questions about associated deficits such as problems in chewing, swallowing, emotional fluctuations, and medication that seemed to affect speech.

The third group included questions about the patients' perception of deficit (i.e., how their speech sounded when the disease began), a description of the current speech difficulty and the consequences of the disorder on the disability/handicap of the patient (if any). In addition, questions about possible voice problems were also included. The final group of questions sought information about the management of the speech problems and the awareness of diagnosis and prognosis by the patient. Other questions that seemed to be relevant in this study were also included. These included information on smoking, drinking, and any medication that may have been taken by the patient before the motor speech examination. Appendix D involves copies of case history forms in the Greek and the English language.

Overall, the history form that was developed for the current study aimed to gather a detailed description of all the information that may be beneficial to the examiner. Apart from the history form, an examination of the speech mechanism, an intelligibility assessment and an acoustic analysis of speech should normally be included in the motor speech examination. In the present study, the examination of the speech mechanism during non speech activities took place through the

administration of the Frenchay Dysarthria Assessment (Enderby, 1983). The intelligibility assessment took the form of a score that reflected an overall judgement of speech ability. The acoustic analysis of speech was replaced by an electrolaryngographic analysis for reasons that will be explained later in this chapter. A description of all these procedures will follow in the next sections.

2.5 The Frenchay Dysarthria Assessment (FDA)

An easily administered dysarthria test in Greek was considered to be appropriate to examine the speech mechanism during some speech, non speech, and voicing activities. However, at present, such a test does not exist in the Greek literature. It was hypothesized that a test translated from the English language to the Greek language could be used. The Frenchay Dysarthria Assessment (FDA) test was constructed in 1980 to fit some presuppositions in the British population: easy application to therapy, easy use and short duration of administration, little requirement for training in its use, and easy communication to other professionals of its results (Enderby, 1980). Although the FDA as a maximum performance test is administered in the English language, its structure and form of administration could be considered "international" because it comprises some (but not all) subtests with non speech, speech, and voicing tasks that could be used in any language. Kent et al. (1987) support the international use of maximum performance tests.

The test is divided into the following sections: reflex, respiration, lips, jaw, palate, laryngeal, tongue, intelligibility, rate, sensation, and associated factors. Each section contains tasks measurable in seconds. In speech pathology, the non speech tasks are considered a major form of discrimination between dysarthria and apraxia of speech (Duffy, 1995). They are also useful to give information about the

absence or presence of neurological diseases including Parkinsonism (Robin et al., 1997) and to show changes before associated changes in perceptually adequate speech occur (see a relevant discussion about the merits and drawbacks of these tests in section 1.7.5). Examples of two non speech tasks, one speech task and one voicing task will follow.

Examples of non speech tasks involve the elevation and lateral movement of tongue that have the patient moving the tongue up and down (outside the mouth towards the nose and chin) or left and right (from side to side of the mouth) five times. The timing of each task determines the subject's score. In the elevation of the tongue "a" is the score for the completion of the task in 6 seconds, "b" within 8 seconds, "c" when tongue moves well but laboriously and incompletely, and so on. A speech task involves the production of vowel combinations [u i][4] ten times to check the function and shape of the lips ("lips" section). Finally, a voicing task involves maximum sustained phonation (prolongation of [a] for a measurable period of 15 seconds[5] to check laryngeal stability ("laryngeal section"). Apart from phonation in the laryngeal area, the FDA also measures intonation (singing a scale) (Leuschel & Docherty, 2000). Considering the similarities of the Greek to English phonology it was hypothesised by the examiner that these tasks can be used in the Greek language. The scoring form of the Frenchay Dysarthria Assessment (FDA) was used and its guidelines were translated into Greek.

As stated above, the administration of the FDA to the Greek population is considered useful because of its easy application, its ability to diagnose dysarthric

[4] "a" score for 10 sec, "b" score for 15 sec, "c" score for laibored movement of the lips, "d" score for different shapes in the "oo ee" production and "e" score for an inability to produce any shape.
[5] "a" score for prolongation in 15 sec, "b" score for prolongattion in 10 sec, "c" score for prolongation in 5-10 sec, "d" score for prolongation in 3-5 sec and "e" score for prolongation in 0-3 sec.

symptoms (timing of the articulators), its short duration and its ability to differentiate the dysarthria among an experimental group (Parkinsonian patients) and a control group (normal population). The FDA was intended to identify dysarthric symptomatology only in one population (idiopathic Parkinsonian patients) and to compare the results with another population (control group). So, it was hypothesised that the FDA could distinguish differences in conditions such as dysarthria compared to non dysarthria. So, it was used not as a differential diagnostic test of dysarthria, as Duffy (1995) commented, but as a diagnostic test per se. The examiner has been trained in the use of the FDA during his graduate studies in United States.

Compared to the other two well known dysarthria tests for the English speaking populations (Robertson Dysarthria Profile and Motor Speech Examination) the FDA includes phonation, loudness and Fo variation in connected speech in addition to maximum performance tasks (Leuschel & Docherty, 2000). Also, although the Robertson Dysarthria Profile includes a more detailed investigation of connected speech and involves intonation and stress patterns in reading, conversation and stress drills, these hardly can be translated into another language (Greek) due to differences between the languages in intonation and stress patterns. The Motor Speech Examination does not contain maximum performance tasks that are considered essential in a study such as this to show changes at the beginning of Parkinson's disease where the subjects expected to be close to normal.

In general, although the FDA has a high intrajudge, interjudge, and test retest reliability in the British population, it is not clear if this stands for the Greek population. A translated test from another language has its own limitations. Stages such as translation and back translation and piloting are important for the

application of tests that are used in another language. Any text has inherent limitations due to social, cultural, historical, racial and sexual factors. Since knowledge in the era of relativism is a human social product (Smith & Deemer, 2003), the importance of society as a factor in the development of new tools is paramount. So, the development of new tools or the translation of tools used in another culture needs to take into consideration these limitations. It is a limitation of the current study that these stages were omitted in the application of the FDA in the Greek language.

However, some precautions have been taken in the application of the translated FDA in Greek to counterbalance some of its limitations. The existence of the control group in this study aimed to counterbalance some of the reliability problems during the administration of a test that has not had yet high intrajudge and interjudge reliability in a language other than English.

The FDA has also received criticisms about its ability to distinguish between different types of dysarthria (Duffy, 1995) and about its ability to evaluate intelligibility (Duffy, 1995; Kent, Weismer, Kent, & Rosenbek, 1989). Duffy (1995) states that there is an overlap in the sections of FDA between different types of dysarthria and a lack of specified criteria to determine each dysarthria type. He concludes that FDA "may be viewed as a test that distinguishes among patients with different lesion loci on the basis of non verbal oral findings and certain speech characteristics, rather than a differential diagnostic test of dysarthria per se" (p. 88). As mentioned above in the present study, FDA was not used as a differential diagnostic test of dysarthria but as a diagnostic test per se, distinguishing between Parkinsonian subjects and control subjects.

Kent et al. (1989) and Duffy criticise the FDA's ability to measure intelligibility overall. They reported that FDA might be beneficial for measuring single word intelligibility but might not be useful for measuring intelligibility in sentences and in connected speech. With regard to the intelligibility estimation, the current study followed Kent's et al. (1989) suggestions to construct an intelligibility test that could possibly be replicated (if standardised) for use in future studies in the Greek population. So, the section on intelligibility of the FDA (repetition, description, conversation) is omitted from the current research as not representative of intelligibility testing and is replaced by a different intelligibility test whose logic will be explained in a following section. Since the sensation and associated factors sections had already been extensively covered in the section on the history form no further coverage was needed.

2.6 The intelligibility testing

This section aims to discuss the concept of intelligibility from different perspectives. Intelligibility is defined and the most preferable method of its estimation in dysarthria is discussed. The following subsections discuss the relationship of intelligibility to phonation and articulation in motor speech disorders, the logic upon which a Greek list of words for intelligibility estimation was constructed for the present study and issues such as familiarisation and judgement that may affect the intelligibility scoring.

According to Schiavetti (1992), intelligibility is defined as "the match between the intention of the speaker and the response of the listener to the speech passed through the transmission system" (p.13). Intelligibility has been used for different purposes in the literature. Some authors have used it as one criterion for the

assessment of the severity of speech disorders in the hearing impaired population (Metz, Schiavetti, & Sitler, 1980) and others have used it as a useful tool in explaining the basis of a speech disorder regarding specific articulatory deficits (Weismer, Kent, Hodge, & Martin, 1988). In other studies, intelligibility has been used as an index of progress in speech therapy with deaf children (Monsen, 1981) and as a functional index of communicative performance (Beukelman & Yorkston, 1979). Hustad, Beukelman and Yorkston (1998) include intelligibility in a broader model of chronic disability that involves assessment in pathophysiology, impairment, functional limitation, disability, and societal limitation. The assessment of functional limitation involves the issue of intelligibility.

Schiavetti (1992) reviewed the different intelligibility measurement methods (scaling procedures vs. word identification tests). The pilot study by Darley et al. (1969a) is an example of a scaling procedure method. In this method the examiner assigns a number that reflects his/her impression of intelligibility severity. Word identification tests involve the examiner to write down or match to similar words the word that he/she listened from the patient. According to Schiavetti, the word identification test is preferable compared to scaling procedures, for many reasons. First, its score is calculated as a percentage of correctly heard words. Therefore, it can be easily used by the speech pathologist and easily communicated to other professionals. Second, it is well associated with the information transfer in dysarthric speakers (Beukelman & Yorkston, 1979). Third, it is easier to administer and score than scaling procedures (Schiavetti, Sitler, Metz, & Houde, 1984). Fourth, there is a good relationship between word identification test and the acoustic characteristics of speech (Kent et al., 1989; Weismer et al., 1988). Along the same lines, Yorkston and Beukelman (1978) compared different techniques for measuring

assessment of the severity of speech disorders in the hearing impaired population (Metz, Schiavetti, & Sitler, 1980) and others have used it as a useful tool in explaining the basis of a speech disorder regarding specific articulatory deficits (Weismer, Kent, Hodge, & Martin, 1988). In other studies, intelligibility has been used as an index of progress in speech therapy with deaf children (Monsen, 1981) and as a functional index of communicative performance (Beukelman & Yorkston, 1979). Hustad, Beukelman and Yorkston (1998) include intelligibility in a broader model of chronic disability that involves assessment in pathophysiology, impairment, functional limitation, disability, and societal limitation. The assessment of functional limitation involves the issue of intelligibility.

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intelligibility of dysarthric speech. Among others, the multiple choice intelligibility scoring technique (as an example of a word identification test) produced the highest score.

Intelligibility is a problematic issue in respect to its definition and assessment method (Connolly, 1986; Kent, 1997). For this reason, a number of different tasks have been used to assess it. Different scores in each task have been found (spontaneous speech, repetition, reading, repeated singing, and spontaneous singing). Kempler and Lancker (2002) report an increased score in reading and a decreased score in spontaneous speaking in a Parkinsonian patient. Also, large differences were found among these tasks ranging from 29% in spontaneous utterances to 88% in spontaneous singing utterances.

The complexity of the issue of intelligibility is due to many factors (Connolly, 1986; Hustad et al., 1998). These include:

- It is a not an all-or-nothing phenomenon but it is a matter of degree that a message can be understood.
- It is inherently context-dependent and varies with the awareness of the listener in the topic of conversation, familiarity with the speaker's voice, and/or accent/dialect.
- It is involved in the broader concept of indeterminability (the condition in which the listener is not able to recover correctly the intended cognitive meaning of an utterance that may be unintelligible, ambiguous, or illusive. Supervenient indeterminability is a special case of indeterminability that is related to the presence of a disorder (Parkinson's disease in the present study).

- Finally, communicative effectiveness (success of the speaker in a social role in specific social situations) is the product of interaction of a number of variables including the intelligibility of speech, the comprehensibility of speech when the context is provided and the complexity and adversity of communication situations.

Other authors include intelligibility to the concept of comprehensibility in which factors pertaining to the speaker and the listener may change the listener's ability to understand the message (Hustad et al., 1998).

In general, intelligibility in the present study functioned as an index of communicative performance in both groups (experimental group before/after medication and control group). More specifically, intelligibility assessment intended to address how well each subject of the Parkinsonian group is understood when talking, compared to a matched control subject and compared to himself/herself after 3-3.5 months of medication. More comments on this will follow in section 2.6.2.

2.6.1 The relationship of intelligibility to phonation and articulation

An intelligibility assessment in every motor speech examination can give an estimation of communicative performance of the subjects. Kent (1988) underlined the importance of intelligibility as an essential feature of speech communication and the assessment of intelligibility as an issue of fundamental clinical importance in dysarthria.

Ramig (1992) reviewed the role of phonation in speech intelligibility. According to her, phonatory disruptions due to neuromuscular malfunctions can reduce speech intelligibility. Her results showed high correlations of monotone and

developed an intelligibility test that aimed to address the above conditions. This test was the framework for the construction of the Greek list for intelligibility and for this reason it will be described in detail.

Kent's test is considered systematic in a sense that it aims to evaluate the "phonetic underpinning of intelligibility deficits" (Weismer & Martin, 1992, p. 69). Kent et al. (1989) grouped words that had a minimal-pair or a near minimal-pair relationship to the target word. The development of their test was based on nineteen acoustic-phonetic contrasts characterised by minimal pairs that are sensitive to dysarthria and could contribute to speech intelligibility. The pairs differed in phonetic dimensions such as voicing, front vs. back vowels, and so forth. According to the authors, this testing could reflect the errors that have been found in different studies in dysarthria. These errors, especially the variables that are focused on imprecise articulation, can possibly have a major impact on speech intelligibility. Kent's test also provides a quantifiable score of intelligibility together with interpretations in terms of the phonetic and acoustic bases of an intelligibility deficit (Weismer & Martin, 1992). Obviously, these interpretations could guide the therapy process if they are well described and defined.

More specifically, Kent's et al. (1989) work is a multiple choice test with a target word and three other words similar to the target word (total number of words was 280). The experimenter records the target words on tape as the patient reads them. The person who is responsible for estimating intelligibility (listener) chooses the word that he/she listened to out of 4 words (that have a minimal-pair relationship to the target word) by listening to the tape. The percentage of correctly heard words is the overall intelligibility score. The selection of the words in this test meets three criteria. First, it allows the examination of several phonetic contrasts as important

dimensions of intelligibility. Second, it represents through the words, as was stated above, phonetic features or contrasts that are vulnerable in the dysarthrias. Third, these phonetic contrasts can be analysed with one or more measures (e.g., acoustics). The concept of random selection of the target words in each tetrad is one of the benefits of the test.

Similar procedures to the work by Kent et al. (1989) were employed in the construction of the intelligibility list of Greek words. This list of words contained the same number of words (280) as in Kent's. Appendices E and F give a description of the Greek word list, the different phonetic contrasts it employed and the similarities/differences between the two tools. The number of parameters for the Greek list of words was found to be double (39) that of Kent's list (19). Differences between the two languages contribute to this difference. Also, three categories in Kent's list with a frequency of occurrence of 8-11 times (alveolar-palatal place contrast, vowel duration contrast, and initial glottal-null contrast) were found not to exist in the Greek language.

The use of a multiple choice intelligibility testing (Kent's list and the Greek list) presents some advantages:

- It is easier to administer. It may take no more than 5 minutes to administer and so, it eliminates/minimises the risk of tiredness in the subject's performance.
- It permits quantification of correct intelligibility percentage.
- It can be used as both a research and a descriptive tool. Although in the present study it may not be relied upon as a descriptive tool, the Greek test could be used in the future in Greece to describe the phonetic

features of dysarthric speech. At the moment, however, where no standardisation process has taken place, the quantification intelligibility estimate is its only purpose.

- As an intelligibility test it is more elaborate and detailed than the intelligibility section of the Frenchay Dysarthria Assessment, which gives intelligibility estimation from only 12 words and 12 sentences.

Compared to the Assessment of Intelligibility in Dysarthric Speakers (AIDS) (Yorkston & Beukelman, 1981a) the Greek word list presents one advantage and one disadvantage. Its advantage is that it does not require construction at the sentence level. Construction at such a level calls for knowledge of other variables (Greek syntax, Greek language complexity) that are difficult to control and match with AIDS. Its disadvantage is that a Greek test similar to AIDS could better estimate the speech rate of dysarthric subjects in addition to the quantification of intelligibility. Again, the existence of no research in Greek language and no reported average rates of speech in the healthy Greek population prohibited the use of AIDS.

An inherent limitation to this method of assessment of intelligibility (word identification testing) is that it involves only information on the adequacy of the speech signal in isolation, ignoring the context, the event, or the purpose of communication. The consequences of this approach may be the underestimation of the functional communication abilities in some dysarthric speakers. Intelligibility measures such as the word lists may not be predictive of real-world communication situations where listeners take into consideration factors such as situation and contextual information in addition to the speech signal (Hustad et al., 1998).

Other factors in intelligibility list of words inherent not to the speech signal but to variables pertaining to the dysarthric speaker (speech supplementation strategies, signalling semantic cues such as topics on a communication board to set the context, and paralinguistic variables such as gestures and facial expressions) or listener (familiarity with the speaker or knowledge of importance to the speaker) may also apply. One study (Crow & Enderby, 1989) that examined some of the aforementioned factors in intelligibility assessment reported an increase to intelligibility of 11% at a word level in Parkinsonian subjects. Also, the issue of having the subject to read test materials (a list of words, sentences, a passage of continuous text, or spontaneous conversation) has its own limitations (Connolly, 1986). This is due to factors such as the change of the speaking style when reading compared to spontaneous speech.

Weaknesses, inherent to its development, prohibit the use of the Greek list to describe articulatory errors. Differences between the Greek language and the English language, no piloting of the Greek list, differences in the frequency of contrasts between sets of words, and differences in the overall number of each phonetic contrast category in each set are enough to distort the results of the test and mislead the examiner if this test would be used for descriptive purposes. Also, no justifiable inferences could be made on the nature of errors that the subjects made, simply because the list does not guarantee that each experimental subject was exposed to the same contrasts in the words provided for reading. In other words, each experimental subject read a different set of words even though this list had a minimal pair relationship to the 3 other sets of words.

However, the Greek list is an early attempt to measure intelligibility in the dysarthric speech and for this reason is used in the present study. Since an

intelligibility test does not exist in the Greek language further refinement of the present list can lead to a more complete intelligibility assessment list. Furthermore, it is based on factors that contribute to dysarthria even though in a different language than the Greek language (Beukelman & Yorkston, 1979; Kent et al., 1989; Weismer et al., 1988; Weismer & Martin, 1992). The selection of 70 words that was administered to each experimental subject was randomly selected as in Kent's list (out of 4 sets of similar sounding words). The number of words of the list and the time that elapsed between the first assessment (before medication) and the second assessment (3-3.5 months after medication) inhibits familiarisation of the experimental subject with the words.

2.6.3 Familiarisation of intelligibility testing and judgement of intelligibility score

This section will discuss issues such as the selection of the listeners for intelligibility estimation and the familiarisation with the intelligibility list. Beukelman and Yorkston (1980) studied the influence of passage familiarity on intelligibility estimates of dysarthric speech among different listeners. Judges included speech pathologists and lay people, all unfamiliar with the specific task for intelligibility. Their results showed that unfamiliar listeners gave lower intelligibility scores compared to speech pathologists. Furthermore, for mild and severely dysarthric speakers, passage familiarity did not influence judges' scores. On the other hand, for the moderately dysarthric speaker, the scores increased markedly with increasing passage familiarity.

In the present study, the intelligibility serves as an index of communicative performance. Two listeners unfamiliar with the intelligibility task were selected for

intelligibility estimation. Both listeners had a university level education in different areas and they were at the same age level (31 years). The judges listened each audiotape and selected 1 word out of 4 that they thought the subject had said (total number of words = 280). Appendix G shows the list of words that was given to the listeners for intelligibility estimation. The listeners were not familiar with the speaker and no contextual cues were given to them. Judge familiarity in the current study seemed not to be an issue of concern because of the nature of the speech sample that gives a random selection of words and because of the fairly big number of words given (70 words).

From the spontaneous conversation between the experimenter and the experimental subjects during the administration of the history form, it was perceived that intelligibility would not be a problem for the particular experimental group. All patients were at the beginning of the disease and the experimenter did not need to ask them to repeat what they said because of possible problems in understanding. The intelligibility scores proved the above hypothesis. Both groups (experimental and control) were judged by the listeners to have intelligibility scores above 90% and no differences in each intelligibility estimation were found between the scores of the judges.

2.7 Passage development

Currently there is no available Greek non standardised or standardised reading passage to provide the "stable" ground for quantitative analyses in motor speech disorders. The need for such a passage, led to a first attempt at its development during this study. The Greek passage is given in Appendix H. A number of parameters were set for the development of this passage:

The first parameter was that the position (initial, medial, and final) of all vowels of the Greek language need to occur in as many as possible different words in the passage. Appendices I and J show the vowels of the Greek language and the combinations of vowels in the words in the reading passage, respectively. The description of the Greek vowels in a triangle in Appendix I was taken from the Aristotle University of Thessalonika, Greece (Instituto Neoellinikon Spoudon, 1996).

Appendix K includes the Greek vowels and consonants in initial, medial, and final position of words included in the reading passage. A short explanation will follow. In the passage, there are 44 words with a vowel in initial position and 12 with the vowel after a consonant in the initial syllable (total number was 56). Out of the 44 words that start with a vowel, in 10 words the vowel stands alone (as a word), in 19 words the vowel is in a V̲CV combination, in 8 words is in a V̲CVC combination, and in the remaining 7 words is in other combinations (V̲C, V̲CVV, V̲CVVC, V̲CVCV, V̲CCV, and V̲CCVC). Out of the 12 words that the vowel follows a consonant in the initial syllable of a word, in 8 words the vowel is in a CV̲CV combination and in the remaining 4 words it is in different combinations (CV̲CVC and CV̲CVCV). The frequency of occurrence of particular vowels was also taken into account but not in a systematic way. For example, the vowel [i] stands alone as an article throughout the text 7 times and the vowel [o] 3 times.

In medial position, there are 17 words where the vowels follow a consonant. The vowel [i] occurs 6 times (4 times in a CV̲C combination), the vowels [u], [o] and [a] occur 3 times each, and the vowel [ε] 2 times, all in different combinations (CV̲C, CCV̲C, CCV̲CV, CVCV̲CV, CVCV̲V, CVCCCV̲CVV, VCV̲C, VCV̲CV̲CVC, and VCV̲CVCCVC). In the final position, there are 26 words where the vowels occur in

different combinations ending with CV. Out of them, 11 words stand in a CV combination and the remaining 15 words in other combinations (CCV, CVCV, CVCVCV, VCV, and VCCV). Finally, there are 5 words in which the final vowels are not in CV combinations (CVCVC, VCCVC, VCVC, VCVV, and VCVCV).

The second parameter was that the position (initial, intervocalic, and final) of all consonants and consonant clusters of the Greek language need to occur in as many as possible different words in the passage (see Appendix L for a list of Greek and English phonemes in the International Phonetic Alphabet).

The third parameter was that the presence of as many as possible diphthongs of the Greek language needs to occur in the passage.

Moreover, attention was given so that the duration of the passage and the total number of its words was as short as possible compared to other passages (Rainbow passage, Arthur the Rat passage). The overall length of the "Greek Islands Passage" consists of 397 words including the headings. In the present study the last paragraph of the passage was omitted to avoid tiredness of the patients. So, the experimental and control group read a total of 316 words. Considering the complexity of the Greek language, the Greek Islands passage has a reasonable length. An alternative to the construction of this passage would be the translation of the Arthur the Rat passage from the English to Greek. This undertaking was attempted and the translated passage consisted of 335 words. However, because in the translated Arthur the Rat passage no controlled parameters existed as in the Greek Islands passage, the latter was preferred for this study.

Finally, the topic of the passage was well known to Greeks and therefore, more easily readable (at least at a semantic level). It involved general geographical and cultural information about the Greek islands, a familiar topic. A small dialogue

was inserted in the passage to reflect different speaking conditions (narrative and dialogue) and inflections of stress. Similar conditions are appeared in other passages ("Arthur the Rat", "Rainbow Passage").

However, the development of a reading passage should satisfy a number of additional criteria, such as:

- Equal representation of the length of words used in the passage
- Involvement of phonotactic patterning (sequencing of sounds)
- Similar frequencies of words in the passage with words in the Greek language.
- A standardisation process.

To compensate for the absence of the above criteria, this passage was administered to both the experimental and the control group. Moreover, its use was limited to the analysis of phonation.

2.8 Usefulness of electrolaryngographic measurement

This section is divided in 3 other subsections and it aims to address concerns during electrolaryngographic measurements, the reliability of the electrolaryngography and the establishment of electrolaryngographic measures in the present study.

Many authors discuss the usefulness of electrolaryngography as well as some of the problems encountered in it (Abberton et al., 1989; Baken, 1992, 1997; Colton & Conture, 1990; Fourcin, 2000; Kitzing, 1990; Titze, 1990). In the eighties, electrolaryngography (ELG) was used more for qualitative purposes (descriptions of laryngeal action) to give a better picture of the performance of the vocal folds especially during the closed phase of vibration.

In the nineties, Baken (1992) discusses that the microphonic effect of the acoustic signal does not influence a significant portion of the electrolaryngogram, that the Lx signal actually represents the surface area of contact of the vocal folds, and that Lx wave can discriminate the three major modes of laryngeal function (normal, breathy, and falsetto). In addition, ELG is reported to be effective as a biofeedback tool in the management of dysphonias together with a display on a oscilloscope screen (Baken, 1997). Furthermore, the Lx signal can be used confidently for the extraction of fundamental frequency (Fo) and jitter measures, less commonly for amplitude-perturbation measurement, but not with amplitude measures and numerical measures (e.g., open quotient) because of ambiguities about the exact instant of glottal opening or closure (Baken, 1992; Colton & Conture, 1990).

2.8.1 Concerns during electrolaryngographic measurement

A number of studies give a comprehensive analysis of electrolaryngography (ELG) characteristics (Baken, 1992; Colton & Conture, 1990; Kitzing, 1990). In this section, some concerns during the ELG measurement will be discussed in the form of instrumental, procedural, and subject/speaking characteristics. Its solutions will be also reviewed.

Instrumentally, the use of automatic gain control and the high-pass frequency characteristics of the ELG may create problems in the interpretation of the output signal. The automatic gain control is used for compensation of variations of the signal level due to varying neck resistance. It has its own time constant to respond rapidly to the changes of the signal level. An increase of its time constant might produce a distortion in the open phase of the signal (Colton & Conture, 1990). This

is an additional reason why many authors support the notion that ELG gives more information on the closed phase of the vocal folds (Abberton & Fourcin, 1984; Baken, 1997). If high pass filtering is not specified in order to interpret the details of the waveform, erroneous conclusions could be reached.

Procedurally, electrode placement, degree of electrode-to-skin contact, and electrode movement during recording might create problems in the output signal. The incorrect placement of the electrodes might give a low signal amplitude. Trial movement of the electrodes up and down along the neck while the subject sustains a vowel will help to overcome this difficulty and to determine the highest possible signal level (Abberton et al., 1989; Colton & Conture, 1990; Kitzing, 1990). Resistance of the skin to the current might also be created because of the properties of human skin. Cleaning the skin with alcohol before the placement of the electrodes will eliminate this resistance. Finally, the movement of the electrodes during the production of running speech can also create problems for the signal even if a flexible neckband is used to keep the electrodes in a stable position on the neck. If the subject holds the electrodes with his/her hands during phonation, this movement might be avoided and a clearer signal will appear.

Subject and speaking task concerns are also reported to create problems in the final output signal. In female voices, less current fluctuation in the signal will take place due to the small size of female vocal folds and due to the large angle of the thyroid cartilages. However, these observations cannot be generalised because there are cases of women where a perfect signal was created and cases of men where the opposite occurred. Colton and Conture (1990) report least usefulness of the electrolaryngographic (ELG) signal in unilateral vocal fold adductor paralysis and in large mass lesions. Unilateral vocal fold adductor paralysis creates an interfold

gap with little or no vocal fold contact and large mass lesions create no complete vocal fold closure. In speaking conditions especially during running speech, low frequency variations might take place due to vertical laryngeal movement. The use of high pass filtering might help to accommodate this problem.

In general, problems in ELG waveform interpretation may also arise due to:

- Vocal fold contact vs. glottal area (not exact points and exact time that vocal fold contact occur)
- The existence of mucus strands (especially during the open phase when mucus might create a path for the current even though the vocal folds are open)
- The relation of the ELG signal to the aspects of vocal fold vibration (the precision in exact time of the opening of the vocal folds). In contrast, vocal fold closure especially in subjects with no obvious voice problem can be determined (Baken, 1992, 1997; Colton & Conture, 1990; Kitzing, 1990).

2.8.2 Reliability of electrolaryngographic measures compared to other techniques

As stated above, one of the criticisms on electrolaryngography (ELG) is its apparent instrumental difficulty to define the precise and appropriate points for measurement especially from the closed phase of the vocal folds to the open phase. However, there are measurements that were proposed to be reliable and useful in a study that aims to investigate patterns of vocal fold vibration. For example, fundamental frequency and its distributions can differentiate normal compared to

abnormal voice characteristics and the simplicity of the electrolaryngographic signal in comparison to the acoustic signal helps to extract the fundamental frequency. The closing time (Qx) as the mean percentage of time that the vocal folds are closed to the total period (measured 70% down from waveform peak) can also be used to give reliable measurements especially in patients with voice problems (see a recent discussion in Fourcin, 2000). In general, the advantages of ELG include accurate measurement of the glottal vibratory period resulting in its use for measurement of intonation contours, the calculation of mean and range of voice fundamental frequency, and the registration/recording of aperiodicities.

More specifically, period measurements, pitch range, fundamental frequency, and the relation of ELG measures to acoustic measures have been discussed. According to Kitzing (1990), electrolaryngography is dependable for the measurement of glottal vibratory period. Periodicity analysis can be used to establish the characteristics of the fundamental frequency of the voice. One recent report emphasises that the fundamental frequency of electrolaryngography is highly correlated to the fundamental frequency of the acoustic signal, $r = 0.99$ (Jiang et al., 1999).

Fourcin (1981, 2000) suggests the use of computerised statistics of period measurements. Numerical data can be extracted and measures of central tendency (mean, median, mode) and range can be shown as a distribution of frequencies (DFx) derived from a period by period basis. The statistics of Fx histograms (mean, median, mode, and range) are correlated with the mean pitch and range of the voice (Kitzing, 1990).

Other reports emphasise the advantages of electrolaryngography (ELG) as compared to acoustic methods. For example, intonation curves based on ELG are

being presented as a string of instantaneous discrete values in contrast to an average estimate as in acoustic fundamental frequency (Abberton & Fourcin, 1997; Fourcin, 1981, 2000; Kitzing, 1990). ELG can also be a very dependable method to show a correlate of perceived harshness or hoarseness through scatterplots (CFx) that show successive periods against each other.

In general, the advantages of electrolaryngography when compared to other techniques are (Baken, 1992; Fourcin, 2000; Kent 1997):

- It generates signals that are not affected by supraglottal influences.
- It is non invasive, innocuous and inexpensive.
- It does not interfere with other variables for example, airflow or glottal area.
- It gives details of aspects of vocal fold function that other techniques are not able to do.
- It is immune to acoustic noise and it can be used in many work environments.

In conclusion, ELG may give us important information about impaired voice quality and possibly about monotonous voice by monitoring the smoothness of fundamental frequency of excitation contour, slope and range, and the distribution of frequencies in the overall fundamental frequency excitation range (Abberton & Fourcin, 1984; Fourcin, 2000).

2.8.3. Establishment of electrolaryngographic measures in the present study

The purpose of this section is to state the factors that are important in voice measurement in neurological disorders and to describe the measures that have been used in other studies together with specific tasks. Finally, based on this discussion the electrolaryngographic measures that have been selected in the present study are outlined.

A number of factors needs to be addressed in a study that aims to investigate voice in populations with a neurological disease. These factors should involve:

- Precise definition of neuropathology.
- Adequate medical and pharmacological information.
- Categorisation of subject groups by stage of disease to reduce heterogeneity.
- Categorisation of subject groups by chronological age.
- Categorisation of subject groups by vocal use.

These factors are considered important for accurate voice measurement and subsequently for the validity of the results (Ramig et al., 1988). The present study took into consideration the aforementioned factors in the selection of the sample.

The majority of studies that used quantitative measures of phonatory function in motor speech disorders employed acoustic methods that involve:

- Measures of central tendency of fundamental frequency (mean, median, or less frequently mode).
- Measures of intensity (less frequently because of difficulties in amplitude control where a sound level meter is required).

- Other measures of voice to examine the open and closed phases of the vocal folds (speed quotient, open quotient, etc.).

These measures are used mostly in sustained phonation. However, there are authors who state that long-term measures of phonatory instability such as the standard deviation of fundamental frequency in sustained phonation (square root of the variance around the mean fundamental frequency) and phonatory measures in connected speech could prove more useful in the extraction of laryngeal dysfuctions in neurological disorders (Kent et al., 1994; Zwirner et al., 1991). Some authors reject specific measures because they found such measures to contribute little to the extraction of laryngeal dysfuctions in neurological disorders. Kent et al. (1994) for example, concluded that there is little evidence that fundamental frequency, jitter and shimmer in sustained phonation have the power to identify speakers with specific neurological disorders. In contrast, other authors state that jitter and shimmer may be useful in clinical diagnosis of laryngeal pathology (Heiberger & Horii, 1982; Horiguchi et al., 1987).

Other researchers favour the assessment of phonetic or voice variation in a combination of tasks such as sustained phonation, reading a passage, and a natural conversation (Brown & Docherty, 1995; Fox & Ramig, 1997; Kent & Kent, 2000). Kent and Kent (2000) add diadochokinesis in the assessment of motor speech disorders. The use of all of the above tasks helps in the classification of different types of dysarthria, in the description of subgroups and individual variations within a dysarthria type, and in the manifestation of pathophysiology in the subsystems of speech production (Kent & Kent, 2000). The employment of all tasks is preferred as a more suitable method because there are indications that

differences in speech behaviour in structured (read material) compared to unstructured tasks (naturalistic conversation) exist. So, the results from structured tasks may be unrepresentative of natural speech behaviour and they cannot be generalised without caution (Brown & Docherty, 1995; Leuschel & Docherty, 2001). Finally, it is during the reading of a passage and during conversation that various prosodic disturbances become evident (Brown & Docherty, 1995; Leuschel & Docherty, 2001). In hypokinetic dysarthria, these disturbances include monoloudness, monopitch, reduced stress, and variable rate with short rushes or accelerated speech.

The present study took into consideration the above findings. Monopitch, primarily, and monoloudness secondarily were chosen to be measured as the most prominent voice features in hypokinetic dysarthria. A detailed quantitative measurement of fundamental frequency, relative intensity, and voice quality in a number of tasks (sustained phonation, reading a passage, and a natural conversation of 3 minutes) took place.

Electrolaryngography (ELG) instead of acoustic methods was used to quantify these aspects of voice. Its advantages and disadvantages were discussed in previous sections. The characteristics of ELG to collect data from the summation of sampling from a connected speech sample and its ability to analyse both tasks (sustained phonation, and reading/conversation) without supraglottal interference (Baken, 1997; Fourcin, 2000) makes it a more favourable instrument in the present study. So, the electrolaryngographic measures that have been used in the present study involve the following:

- Mean Fx (fundamental frequency of excitation derived from the measurement of the time distance, Tx, between successive epochs of excitation), and standard deviation Fx.
- 90% Fx range as the frequency values above and below the average between which 90% of the observed frequencies fall.
- Mean Ax (as mean relative intensities measured from the correspondent to Lx acoustic peak in each acoustic period), standard deviation Ax, and 90% Ax range.
- Jitter and shimmer.
- Average Qx, standard deviation Qx, and Qx range (maximal Qx-minimal Qx).

In the current study, possible correlates of monopitch and monoloudness as the primary features of hypokinetic dysarthria were proposed. Table 3 at the end of this section, explains the proposed interrelationships of the laryngeal pathophysiology in idiopathic Parkinson's disease (PD), the perceptual characteristics of speech and the electrolaryngographic variables for measurement in this study. In reading and conversation both first and second order distributions were measured (DFx1, DFx2, DAx1, DAx2) but for reasons of clarity they are excluded from table 3. However, Fx denotes both DFx1 and DFx2 (first and second order of fundamental frequency) and the same occurs for relative intensity (for a description of these measures, see the relevant section in literature review). The mean and the standard deviation (SD) of the above proposed measures were calculated. Also the ranges of fundamental frequency, relative intensity, and the ratio of time during the closed phase divided by the total period (measured 70%

down of the waveform peak) were measured. In Figure 17, a list of all tasks and the corresponding fundamental frequency variables are shown.

Sustained Phonation	Reading	Conversation
- Average Fx	- Mean DFx1	- Mean DFx1
- SDFx	- SDDFx1	- SDDFx1
- Jitter	- 90% Range DFx1	- 90% Range DFx1
	- Mean DFx2	- Mean DFx2
	- SDDFx2	- SDDFx2
	- 90% Range DFx2	- 90% Range DFx2

Figure 17. Fundamental frequency variables used in the study in all tasks.

Figure 18 shows a list of all tasks and the corresponding relative intensity variables. Relative intensity is derived from the corresponding to Lx period acoustic peak in each acoustic signal period (Fourcin, 2000). Caution, however, should be taken in the interpretation of the results in relative intensity because of a lack of a sound level meter. However, all the necessary precautions were taken to insure the better collection of intensity measurements.

Sustained Phonation	Reading	Conversation
- Shimmer	- Mean DAx1	- Mean DAx1
	- SDDAx1	- SDDAx1
	- 90% Range DAx1	- 90% Range DAx1
	- Mean DAx2	- Mean DAx2
	- SDDAx2	- SDDAx2
	- 90% Range DAx2	- 90% Range DAx2

Figure 18. Relative intensity variables used in the study in all tasks.

Figure 19 below shows the closed period of excitation variables/contact phase ratios (DQx) that have been used in the current study in the different tasks.

Sustained Phonation	Reading	Conversation
- Average Qx	- Mean DQx1	- Mean DQx1
- SDQx	- SDDQx1	- SDDQx1
- Qx Range	- 90% Range DQx1	- 90% Range DQx1

Figure 19. Closed periods of excitation variables used in the study in all tasks.

Table 3. Hypothesised interaction of pathophysiology, perceptual speech characteristics and electrolaryngographic measures in Parkinson's disease

Laryngeal Pathophysiology	Perceptual Characteristics	ELG Variables Measured		
		Sustained Phonation	Reading	Conversation
1. Bowed vocal folds, rigidity, hypokinesia in the laryngeal muscles (Critchley, 1981; Hanson et al., 1983)	Reduced loudness, weak voice (Baker et al., 1998; Fox & Ramig, 1997; Logemann et al., 1978)	• Shimmer	• Mean Ax • St. Deviation Ax • Ax Range	• Mean Ax • St. Deviation Ax • Ax Range
2. Rigidity of the cricothyroid muscle and thyroarytenoid muscle (Aronson, 1990; Gallena et al., 2001)	Reduced pitch variability, monopitch (Darley et al., 1969a, 1969b, 1975; Gentil & Pollak, 1995)	• Jitter	• Mean Fx • St. Deviation Fx • Fx Range	• Mean Fx • St. Deviation Fx • Fx Range
	Breathy voice quality (Chenery et al., 1988; Ludlow & Bassich, 1983; Zwirner & Barnes, 1992; Gallena et al., 2001)		• Qx • St. Deviation Qx • Qx Range	• Qx • St. Deviation Qx • Qx Range

CHAPTER 3. METHODOLOGY

This study examines the following questions:

- Do patients who have been diagnosed with early idiopathic Parkinson's disease (PD) exhibit dysarthria?
- Are their speech and aspects of voice different from the speech of people without a neurological disease?
- Does medication affect their speech and aspects of voice?

This chapter describes the research design, the subjects and the procedure of the current study.

3.1 The research design

Descriptive research is used in behavioural sciences to examine group differences and/or group relationships. By definition, descriptive research does not make cause-effect inferences because other factors inherent to human nature (e.g., psychological factors, family environment), may be responsible for the results rather than the independent variable (classification variable). Comparative research is also used very often in the field of Speech and Language Pathology to observe speech differences, whereby two or more types of groups are compared to make conclusions about their differences or similarities. Ideally, a large number of subjects in descriptive comparative studies help the experimenter to make more valid conclusions in contrast to a study that employs a smaller number of subjects. However, there are some studies where this is not possible. Studies that aim to investigate in depth the differences between medicated/unmedicated persons with neuromuscular disorders usually employ a small number of subjects because other factors (e.g., type and time of medication, variability in the effects of the disease) prohibit adequate matching

between groups. Also, the organisation of the health system itself may raise difficulties in finding subjects that have not been treated before. For example, in the present study a decision had to be made, due to specificities of the Greek health system (similar to the English health system), either to employ a large number of unmedicated subjects with a high degree of misdiagnosis (see a relevant discussion by Hughes et al., 1992), or a smaller sample size with a more definite neurological diagnosis.

The current study is a descriptive comparative study. A Parkinsonian group (before medication and 3-3.5 months after medication) and a matched (in age, sex and education) control group were compared in terms of their speech and phonatory ability (criterion or dependent variable). This study examined the speech symptomatology of subjects in the beginning of Parkinson's disease in an outpatient university clinic over an eighteen-month period.

The planned research design of this study was a pretest-posttest matched descriptive design in which the two groups would be measured at two specific times. Because of the mortality effects that took place after the pre-test, especially in the control group (almost half of the subjects either left the study or was not possible to locate them), this design could not be used. Instead, a different research design was chosen for each experimental question.

Experimental Questions 1 and 2: This is a between group research design in which the two groups (one Parkinsonian and one control group) were measured once. The classification variable (independent variable) that was manipulated in these experimental questions was Parkinson's disease and the criterion variables (dependent variables) were aspects of voice (fundamental frequency, relative intensity, voice quality) and speech (Frenchay dysarthria assessment scoring and intelligibility scoring). However, due to the small size of the

experimental group it was decided to employ a methodology, which looks at both individual and group analyses. With the group analysis statistical comparisons of the averages in different groups form the basis for conclusions. However, the size of the sample makes these comparisons vulnerable to individual extreme scores and limits their generalizability. Therefore, observation of the individual data was judged as necessary in order to identify possible tendencies or generate hypotheses that need to be tested in future research. Question 1 was used as a general question that involved the inferences of the findings in question 2 (speech and intelligibility).

Experimental Question 3: This is a within - group research design in which one group (mildly dysarthric Parkinsonian group) was measured at a specific time and it was re-measured after 3-3.5 months of medication. The classification variable in this experimental question was medication and the criterion variable was speech and a number of aspects of voice (as for experimental question 2). Inferences about differences in the criterion variable were made. As in experimental question 2 due to the small size of the sample, a methodology that looked at both individual and group analyses took place.

3.2 Subjects

3.2.1 Subjects–Experimental group

The exclusion criteria that were employed in this study have been analysed in detail in chapter 2. Briefly, these involved:

- The appearance of speech and language problems during childhood
- The existence of laryngeal pathology at the time of speech assessment
- The levodopa medication at the time of diagnosis

- Visual and hearing problems severe enough to interfere with reading and listening
- Illiteracy
- Other neurological problems and cognitive problems as measured by the Mini Mental State Examination (MMSE) (Folstein et al., 1975).

The experimental subjects in this study were diagnosed with Parkinson's disease a few hours before the speech assessment and exhibited bradykinesia and tremor as neurological signs. All subjects were found to be at stage 1 on the Hoehn and Yahr scale. Two neurologists specialising in Parkinson's disease in the outpatient university neurological clinic of the "Aiginitio Hospital" of Athens and in the neurological clinic of the General Peripheral Hospital of Athens "Georgios Gennimatas" made the diagnosis. Two of the experimental subjects (KP6, GEI13) were diagnosed with Parkinson's disease in the General Peripheral Hospital of Athens "Georgios Gennimatas". The remaining experimental subjects were diagnosed with Parkinson's disease in the outpatient university clinic in "Aiginitio Hospital" of Athens.

Sixteen subjects (10 males and 6 females) were found in a period of 18 months. From this initial group, 13 subjects (8 males and 5 females) took part in the study. The subjects ranged in age from 43-80 years (mean age 63 years). Their education ranged from 4-24 years (mean education time 11.8 years). The different medication (no levodopa) of 2 subjects (KP6, GEI13) compared to the other 11 subjects reflects the different clinical approach of the consultant neurologists. The general profile of the 13 experimental subjects is listed in Table 4.

Table 4. Experimental subjects' biographical information.

SUBJECTS	SEX	DOB – AGE	SMOKING PER DAY	L-DOPA MEDICATION BEFORE DIAGNOSIS	L-DOPA MEDICATION AFTER DIAGNOSIS	EDUCATION
TZS2	F	6/2/1935 **64,8**	No	No	-Akineton 1×3 -Madopar 1×3 -Symmetrel 1×3	5 years
KD4	M	30/10/1933 **66,1**	No	No	-Madopar 1×3 -Akineton ½×2 -Symmetrel 1×2	18 years (Univ. level)
SB5	M	2/1/1921 **79,9**	No	No	-Madopar 1×3 Stopped after 2months of medication	16 years (Univ. level)
KP6	M	7/1/1937 **62,10**	No	No	-Requip 2×3	24 years (Univ. level)
PM7	M	17/12/1934 **65,1**	No	Akineton Sinemet Symmetrel[6]	-Madopar 1.5×3 -Akineton 1×3	6 years
BI8	M	7/2/1926 **74,2**	4	Akineton 1×2	-Madopar ¼×3 -Akineton 1×1	12 years
PX9	F	18/9/1936 **63,8**	No	No	-Madopar 1×3 -Akineton ½×3	9 years
SI10	M	6/2/1946 **54,4**	30	No	-Madopar 1×3 -Akineton 1×3	6 years
GN11	M	23/3/1936 **64,3**	No	No	-Madopar 1×3 -Akineton ½×3	12 years
GEI13	F	28/3/1956 **44,4**	No	No	-Mirapex 1×3	14 years
LI14	M	27/8/1957 **42,10**	No	No	-Madopar ½×3 -Mirapex ½×3	16 years (Univ. level)
KA15	F	15/1/1935 **65,9**	No	No	-Madopar 1×3 -Akineton 1×3	4 years
MD16	F	15/12/1927 **72,11**	No	No	-Madopar 1×3 -Akineton 1×3	12 years

So, the final number of subjects enrolled in the present study was thirteen. One subject (PX9) reported laryngeal pathology due to surgery and it was decided to exclude her from the between group results. A group of 8 Parkinsonian subjects scored lower in the Frenchay Dysarthria Assessment (FDA) and were characterised as mildly dysarthrics. The intelligibility assessment did not further differentiate between the two groups. This group of eight mildly dysarthric subjects was used for the electrolaryngographic (ELG) analysis. However, in one subject (KA15) it was not possible to establish ELG

[6] Stopped six months ago

signal due to fatty neck tissue. Then this subject left the study. Finally, another subject (SB5) in the group of mildly dysarthrics, stopped taking medication and for this reason it was decided to treat his ELG results separately.

The mildly dysarthric subjects were used to examine the effect of medication. As mentioned before, one subject (KA15) left the study and another subject (SB5) was treated as a separate case. Finally, the subject with the laryngeal pathology (PX9) was included in the within group results, to observe the effect of medication especially on the FDA, and then on the intelligibility assessment and ELG measurement. In the beginning of chapter 4 and 5, more details are given regarding the numbers of subjects and the rationale for their inclusion or exclusion. Table 5 below explains who left the study, who remained and who had medication.

Table 5. Subject participation in the present study

Subjects	KD4 Male	SB5 Male	KP6 Male	PM7 Male	BI8 Male	SI10 Male	GN11 Male	LI14 Male	TZS2 Female	GEI13 Female	KA15 Female	MD16 Female	PX9 Female
BETWEEN GROUPS													
FDA scoring	MD	MD	Normal	MD	MD	MD	Normal	Normal	MD	MD	MD	Normal	MD not included due to laryngeal pathology
Intelligibility	NORMAL ABOVE 95% IN INTELLIGIBILITY												
ELG	+	Separate Case (stopped medication)		+	+	+			+	+	No ELG Signal		
WITHIN GROUP													
FDA scoring	+			+	+	+			+	+	Left Study		+
Intelligibility	+			+	+	+			+	+			+
ELG	+			+	+	+			+	+			+

MD = Mildly Dysarthric, Participation = symbol (+)

3.2.2 Subjects–Control group

Thirteen control subjects were matched to the experimental subjects in age, gender and education. The control subjects ranged in age from 44-78.5 years (mean age 63.6 years). Their education ranged from 4-24 years (mean education time 13 years). The general profile of the control subjects is listed in Table 6 (in order of pair matching with the experimental subjects). The same exclusion criteria as for the experimental group were used in the control group.

Table 6. Control subjects' biographical information

SUBJECTS	SEX	DOB - AGE	SMOKING PER DAY	EDUCATION
BA3	F	11/1936 **63,6**	No	4 years
BE1	M	14/3/1932 **67,10**	No	24 years (univ. level)
BI6	M	13/1/1922 **78,5**	No	12 years
NP13	M	22/11/1935 **65**	5	24 years (univ. level)
KP10	M	2/4/1933 **67,10**	No	4 years
AN2	M	20/8/1925 **74,9**	No	12 years
AA9	F	23/6/1937 **63**	No	13 years
SB7	M	18/6/1945 **55**	No	12 years
PK4	M	26/1/1936 **64,4**	30	13 years
THM5	F	31/10/1956 **44,4**	No	19 years (Univ. level)
PN12	M	9/12/1954 **45,11**	No	15 years (univ. level)
PE11	F	16/2/1933 **67,9**	No	6 years
GD14	F	22/7/1929 **71,4**	No	14 years (univ. level)

3.3 Research procedure

As soon as the neurological diagnosis had taken place, the patients were informed about the scope of the study and they agreed to take part. Following this, they signed a consent form and agreed to delay their medication for 48 hours in order to participate in the study. Then a motor speech examination took place involving:

- A history form for speech and voice and the administration of the Mini Mental State Examination (MMSE) in the first meeting,
- The Frenchay Dysarthria Assessment (FDA) and an intelligibility assessment in the second meeting, and
- Electrolaryngographic measurement in sustained phonation, reading of a passage and a conversation in the third meeting.

The intelligibility assessment and the whole of the third meeting were tape-recorded.

The first meeting took place in the hospital immediately after the neurological diagnosis. A history form was administered to the patients and/or relatives (when the patients were accompanied by relatives). As stated in chapter 2, in the history form critical information involved:

- Developmental speech and language problems
- Speech problems
- Voice problems
- Dysphagia problems
- Smoking and drinking information.

The Mini Mental State Examination (MMSE) was then administered and the subjects with a score of 24 and above were judged to have normal cognitive functions.

At the second meeting that took place in the patient's home, the Frenchay Dysarthria Assessment (FDA) was administered. The sections of Reflex, Respiration, Lips, Jaw, Palate, Laryngeal and Tongue were administered according to the instructions included in the assessment (Enderby, 1983). As mentioned in chapter 2, the intelligibility section of the FDA was omitted. Similarly, the "Influencing Factors" section was omitted from the assessment because this information had been collected through the history form.

After the administration of the FDA, the intelligibility assessment took place and was tape-recorded. A list of 4 sets of words (70 Greek words in each set), a total of 280 words, was constructed based on the work by Kent et al. (1989). Each word was typed on a flash card with big letters (0.5 cm tall) to facilitate reading. The subject randomly picked one out of the 4 sets of cards (A, B, C, D). The examiner presented a card to the subject at a rate of one per two seconds, and the subject read each of the 70 cards aloud.

The subject sat on a chair in front of a table and the examiner sat on the right side of the subject to control the volume of the recording. A CP 430 Marrantz Model tape recorder was used, connected to mains power. A Marrantz EM-8 stereo microphone was placed about 15-20 cm from the mouth of the subject on a boom stand on a table. All the recordings took place using TDK SA 60 or 90 chromium dioxide tapes to ensure as far as possible a high quality tape recording. The subject was asked to count twice from 1-10 in order for the examiner to adjust the volume of the recording (the needle on the VU meter was positioned in the midrange of the dial). The following instructions were given before each recording:

" I want you to read some words from the cards. Each card has one word. Some of the words that you will read have no meaning but try to read

whatever you see. Please, read as we speak together naturally, not loudly and not whispery".

During the recording, verbal reinforcement was given (eg. "you are doing well"). All the recordings were made in the morning or early afternoon to ensure that the subjects were given a rest. After the recording the examiner thanked the subject for his/her help and arranged an appointment for the third meeting on the following day. At the end of the meeting the tape was marked with the initials of the subject, the number of the subject and the date. During the second and third meetings, recording protocols were filled out (Appendix M).

Two independent listeners listened to the tapes using the same tape recorder (Marrantz CP 430) and a set of stereo dynamic AIWA HP-X705 headphones. Each listener received a list of the 280 words (all sets). They selected the word they believed to have been spoken in the recording from a choice of four words (the target word and three similar words). The listeners could choose to repeat the recorded words as many times as they wanted.

After this, the examiner scored each list from each listener using the following procedure. The number of correct words divided by 70 (the total number of words) and multiplied by 100 represented the subject's percentage intelligibility for that listener. The average of the two listeners was the final intelligibility score of the subject. The research practice showed that the listeners' intelligibility scores did not differ by more than 2-3%. The criterion level of the intelligibility score was established at 90%[7]. No subject with an intelligibility score below 90% was found, possibly because the subjects were at the very beginning

[7] This study did not aim to measure articulation. Some of the tasks in the motor speech examination will be used in another study to measure articulation with acoustic measures. The criterion of 90% intelligibility serves this logic. The criterion of 80% intelligibility has been used in some studies to ensure that intelligibility was not a major factor in the acoustical measures (Caruso & Burton, 1987; Seikel, Wilcox, & Davis, 1991).

of the disease and no worsening of their physical abilities was evident except from their clinical symptoms during the neurological examination. There was no clear information about how long each subject had had the symptoms that sent them to the neurologist. Only two subjects reported that "they did not feel their hand" for almost a year prior to going to the neurologist. This is a common problem in Parkinson's disease in which it is likely that the preclinical phase can extend years before the patients go for their first neurological examination.

The third meeting took place in the office of the examiner one day after the second meeting. All recordings took place in the morning except where this was impossible because the subject was working in the morning. In this case, the meeting took place in the afternoon and the same time was used for the meeting with the pair-matched control subject. The subject was seated on a chair in front of a table and the examiner sat to their left to control the level of the recordings. Before the recordings, the examiner explained to each subject the use of electrolaryngography. The examiner made a trial recording of himself in order to make the subject feel more comfortable with the instrument.

The recordings were made using a portable TCD-D8 SONY DAT tape recorder that was connected to a electrolaryngograph processor (PCLX) and a Thandar SC110 portable oscilloscope. The oscilloscope was used to offer visual representation of the glottal signal. One set of electrodes connected to the electrolaryngographic processor was used to trace the glottal signal. All three instruments were connected to each other and to mains power. A set of headphones was connected to the DAT tape recorder and used by the examiner to control the quality of the recordings. A blank digital audio tape SONY 60 or 90 was used for each recording. Figure 20, taken from Carlson (1995), shows the recording and processing system that was used.

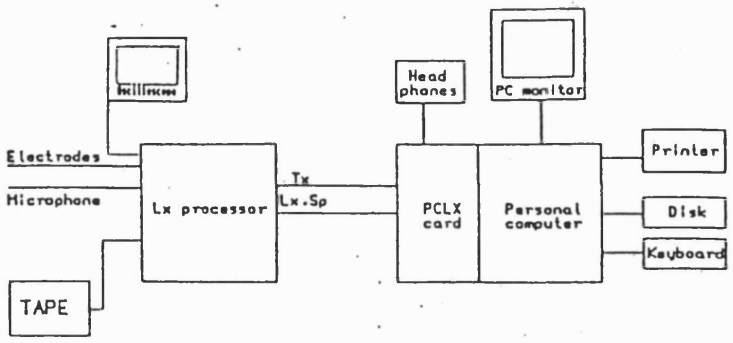

Figure 20. The PCLX system used in the present study.

The examiner pushed the recording and pause button. Then he put the electrodes on the thyroid alae of his neck while phonating [a] showing the signal that appeared in the oscilloscope screen. In this way, the subjects were visually familiarised with the signal and the instrument. The proper position of the electrodes was also explained to each subject. It was also emphasised that it might be difficult to place them exactly on their thyroid wings to ensure that the subjects would be patient during the procedure. Following this, the electrodes were set and held with an elastic band on the thyroid wings of the larynx of the subject while he/she sustained the vowel [a] to ensure that the oscilloscopic signal was strong and to ensure that the processor was receiving the voice signal. Then, the examiner controlled the recording level by asking the subject to count from 1-10 twice. Finally, the experimenter released the pause button to record Alternating Motion Rates (AMRs), sustained phonation of each Greek vowel, reading of the Greek passage and conversation with the subject on a topic (the effect of earthquakes on the subject's life) for 3 minutes. A detailed description of the recording procedure follows.

First, the subject was asked to produce AMRs. The examiner gave the following instructions:

"The first thing I'll ask you to do is to repeat as fast as you can /papapa/ until I'll tell you to stop".

The same instructions were given for /tatata/, /kakaka/ and /pataka/.

Following this, the subjects were asked to sustain each vowel of the Greek language twice for approximately 3-4 seconds at a comfortable pitch as steadily as possible. The examiner gave specific instructions:

"Now, I am going to ask you to do sustained phonation. In other words, to hold an [a] without a change of pitch".

The examiner demonstrated to each subject how to sustain a vowel. When the subject finished this task, the examiner asked him/her to read the Greek passage.

"Now, we are going to read a passage. It is a story that has most of the sounds of the Greek language".

At the end of the reading of the passage the examiner asked each subject to converse about a common topic:

"Now, we are going to talk a little bit about the earthquake that happened a year ago. What was your experience of it?"

Because an earthquake occurred in Athens a year ago, it was judged that all subjects would be interested to express their ideas of where they were when the earthquake struck, what they did at that time and how they reacted to it. The majority of the subjects were eager to talk about this topic and most of them extended the recording time to 5-10 minutes.

No subject seemed to exhibit discomfort or tiredness during the session. At the end of the session the examiner marked the tape with the subject's initials, number and date, and thanked them for their patience. All subjects were reminded that the experimenter would carry out the same procedures after 3-3.5

months in order to see any changes in their speech and voice after taking medication. The same procedures were followed for the control subjects.

After 3-3.5 months the examiner called the experimental subjects to set up a meeting for a new recording. At the beginning of the first meeting the examiner talked with the subjects about their general condition and if they felt there had been any change in their speech and voice because of the medication. They were also asked what their current medication state (type and dosage) was. Subjects reported no other medical problems. The same standard research procedure that was followed before medication, was followed after medication (including the MMSE, the Frenchay Dysarthria Assessment and the recordings).

The DAT tapes were analysed with a computer program (Speech Studio) that digitised the recorded electrolaryngographic signals at a sampling rate of 20 KHz and saved the data as a computer file.

The electrolaryngographic signals were low pass filtered as indicated by the instrument's specifications and displayed on the computer monitor. The experimenter located the points for the demarcation measures for sustained phonation. A one second segment in the middle of the signal (steadiest airflow) was selected for analysis. The electrolaryngographic analysis of the one second segment was derived from the second trial. A similar analysis was made for the reading of the Greek passage (demarcation points in the beginning of the passage and the end of the passage) and conversation (demarcation points from the beginning of the patient's monologue to the end of the third minute).

CHAPTER 4. RESULTS BETWEEN THE PARKINSONIAN AND THE CONTROL GROUP

For reasons of simplicity, the results of the present study are divided in two chapters. Chapter 4 compares the results between the experimental group (Parkinsonian group before medication) and the control group while chapter 5 compares the results between the experimental group before and after medication. The structure of the results in both chapters follows the order of the speech assessment:

- The results of the Frenchay Dysarthria Assessment (FDA)
- The results of the intelligibility testing
- The electrolaryngographic (ELG) results

As mentioned in the methodology chapter (see table 5) the final number of subjects enrolled in the present study was thirteen. One subject (PX9) reported laryngeal pathology because of surgery of the parathyroid glands and one thyroid gland in 1990. In the present study the voice of this subject was perceived as harsh and for this reason it was decided to exclude her from the between group analysis in order not to confound the between group results. Since harshness in this subject's voice may be due to either Parkinson's disease or surgery, the comparison of this subject with her matched pair control would have given questionable results. So, in this chapter the results of the twelve remaining subjects are presented and analysed. The Frenchay dysarthria assessment gave a group of 8 mildly dysarthric Parkinsonian subjects when compared with their matched pair controls. The intelligibility assessment did not further differentiate between the groups. All Parkinsonian subjects scored 95% and above. This group of 8 mildly

dysarthric subjects was used for the electrolaryngographic (ELG) analysis. However, in one subject (KA15) it was not possible to establish ELG signal due to fatty neck tissue. This subject then left the study. Finally, another subject (SB5) stopped taking medication two months after its initiation and for this reason he was treated as a separate case and his results are presented in chapter five. So, the number of subjects whose ELG results are analysed in this chapter is six.

4.1 Results of the Frenchay Dysarthria Assessment

The Frenchay Dysarthria Assessment (FDA) showed the areas where the experimental group scored lower than the control group. The sections of the FDA where differences were found of at least a scale (i.e., a to b) are shown below in table 7. Reflex and palate were omitted from the table because of their scarcity of appearance in the results. However, their results were presented in the individual figures of the matched pairs of subjects, where necessary. No differences in all subjects were found in the areas of respiration and jaw.

In table 7, the following symbols were used:

- A positive sign (+) when the experimental subjects scored lower than the controls
- A negative sign (-) when the experimental subjects scored higher than the controls
- An equal sign (=) when no difference was found between the experimental subjects and the controls.

Only one control subject (GD13) scored lower than its corresponding experimental subject (MD16) did. Probably this occurred because the control subject reported spastic bronchitis at the time that the dysarthria testing took place.

Overall, eight of the twelve experimental subjects (KD4, SB5, PM7, BI8, SI10, TZS2, GEI13, KA15) were found to have lower scores than their matched controls. The areas that were mostly affected in the dysarthria assessment were the tongue area (6 positive signs) followed by the laryngeal area (5 positive signs) and the lips area (4 positive signs). Copies of the completed Frenchay Dysarthria Assessment forms of all subjects are shown in Appendix N.

Table 7. Differences between the Parkinsonian group and the matched pair control group in the Frenchay Dysarthria Assessment.

Subjects	Lips	Laryngeal	Tongue
Males			
KD4 vs. BE1	+	+	+
SB5 vs. BI6	=	=	+
KP6 vs. NP12	No difference		
PM7 vs. KP9	+	=	=
BI8 vs. AN2	+	+	+
SI10 vs. SB7	=	=	+
GN11 vs. PK4	No difference		
LI14 vs. PN11	No difference		
Females			
TZS2 vs. BA3	+	+	+
GEI13 vs. THM5	=	+	=
KA15 vs. PE10	=	+	+
MD16 vs. GD13	=	-	=

The results of the subjects in the Frenchay Dysarthria Assessment (FDA) are presented in an order of involvement (the subjects that showed involvement in more

areas of the FDA are presented first). Figures 21-25 show the results of the FDA for the subjects who revealed differences compared to their controls in more than one area of the FDA. One experimental subject (TZS2) exhibited lower scores than its matched pair in 4 areas of the FDA (lips, palate, laryngeal and tongue). Figure 21 below shows the areas that TZS2 differed from its matched pair control subject BA3.

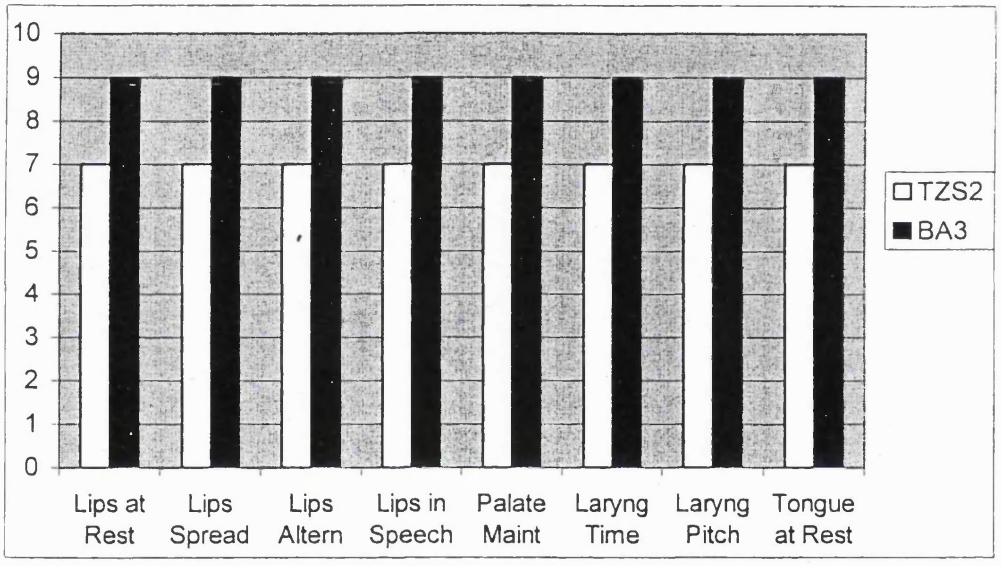

Figure 21. Results of the Frenchay Dysarthria Assessment in the matched pair of subjects TZS2 and BA3.

Three experimental subjects (BI8, KD4, KA15) exhibited lower scores than their matched pairs in 3 areas of the FDA. BI8 and KD4 scored lower in lips, laryngeal and tongue while KA15 scored lower in reflex, laryngeal and tongue. Figures 22, 23 and 24 below show the areas that BI8, KD4 and KA15 differed from their corresponding matched pair controls.

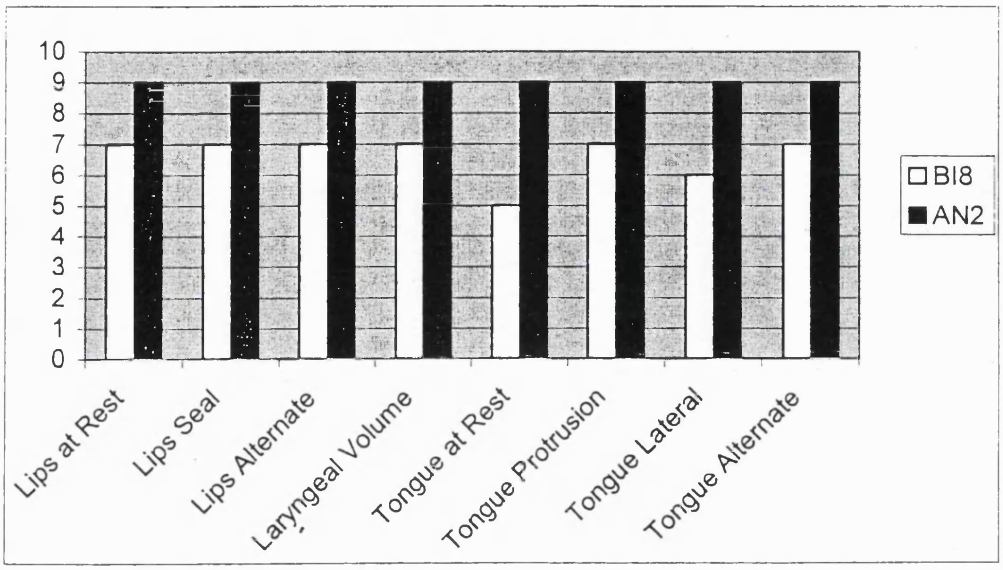

Figure 22. Results of the Frenchay Dysarthria Assessment in the matched pair of subjects BI8 and AN2.

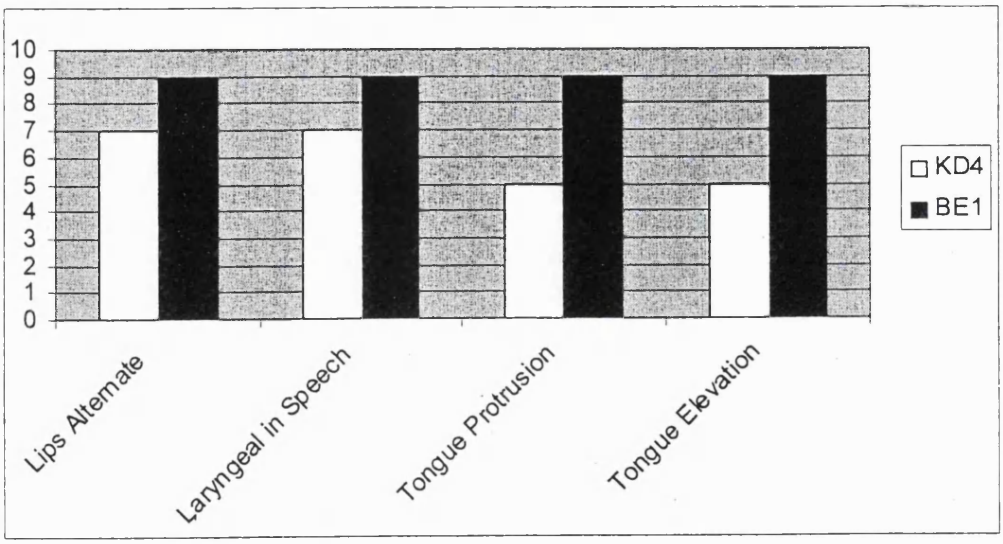

Figure 23. Results of the Frenchay Dysarthria Assessment in the matched pair of subjects KD4 and BE1.

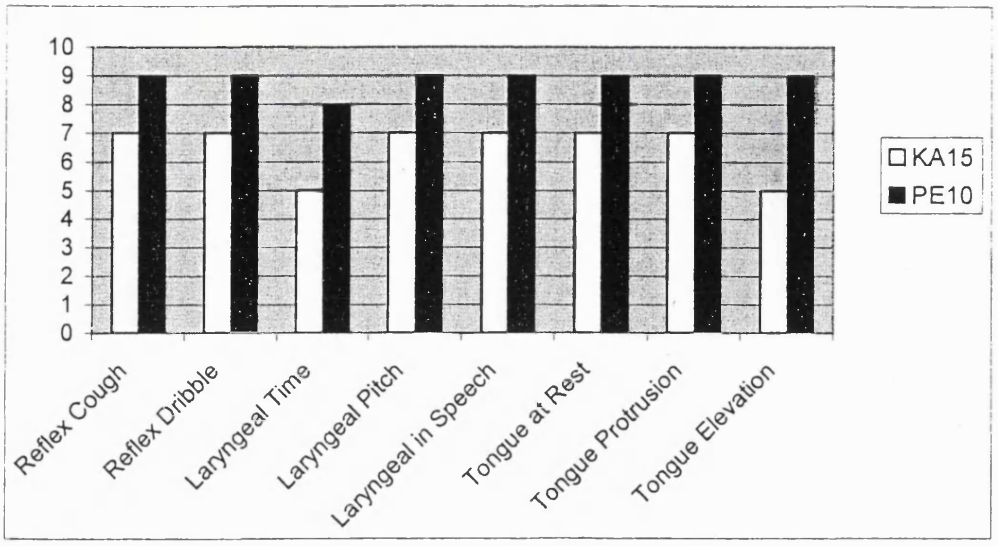

Figure 24. Results of the Frenchay Dysarthria Assessment in the matched pair of subjects KA15 and PE10.

One experimental subject (PM7) exhibited lower scores than its matched pair in two areas of the FDA (reflex and lips). Figure 25 below, shows the areas that PM7 differed from its matched pair control subject (KP9).

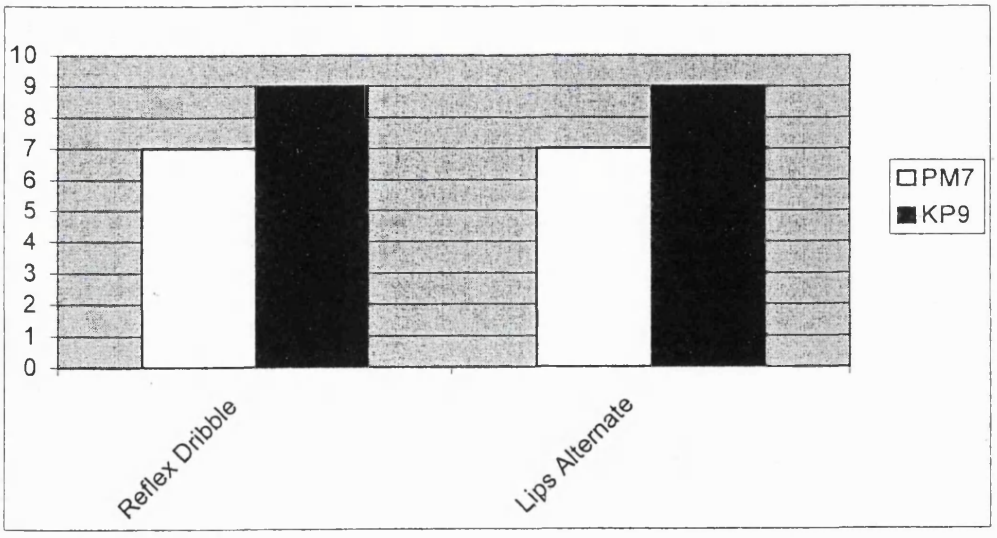

Figure 25. Results of the Frenchay Dysarthria Assessment in the matched pair of subjects PM7 and KP9.

Finally, three experimental subjects (SB5, SI10, GEI13) exhibited lower scores in one area of the Frenchay Dysarthria Assessment. SB5 and SI10 scored lower in the area of tongue (elevation and protrusion) while GEI13 scored lower in the area of laryngeal (laryngeal time).

4.2 Intelligibility results

The intelligibility scores of the Parkinsonian and the control groups are shown below in table 8. All intelligibility scores are in the form of a percentage. The total number of correctly heard words that were identified by the two listeners was divided by 140 (70 words in each listener, total words = 140) to give a proportion of correct words for each subject. This proportion was multiplied by 100 to give the average percentage of correct words pronounced by the subjects in the intelligibility testing. The parentheses below the intelligibility percentage show the average number of words pronounced correctly by each subject.

Table 8. Intelligibility scores between the Parkinsonian group and the matched pair control group.

Experimental Subjects	Intelligibility Scoring	Control Subjects	Intelligibility Scoring
TZS2	96.42% (67.5/70)	BA3	94.28% (66/70)
KD4	98.57% (69/70)	BE1	100.00% (70/70)
SB5	98.57% (69/70)	BI6	99.28% (69.5/70)
KP6	99.28% (69.5/70)	NP12	97.14% (68/70)
PM7	95.00 (66.5/70)	KP9	98.57% (69/70)
BI8	98.57% (69/70)	AN2	97.85% (68.5/70)
SI10	96.42% (67.5/70)	SB7	98.57% (69/70)
GN11	100.00% (70/70)	PK4	100.00% (70/70)
GEI13	97.14% (68/70)	THM5	98.57% (69/70)
LI14	98.57% (69/70)	PN11	99.28% (69.5/70)
KA15	95.71 (68/70)	PE10	91.42% (64/70)
MD16	98.57% (69/70)	GD13	97.14% (68/70)
Group Mean	97.68%	Group Mean	97.67%
Standard Deviation	1.62	Standard Deviation	2.52

The Mann-Whitney test was used to identify differences in intelligibility between the group of the 12 Parkinsonian subjects and the group of their matched pair controls. The results of Mann Whitney U analysis indicated no significant between group differences in intelligibility (Mann Whitney U = 63.5, Z = -0.501, $p > 0.05$). Both groups had the same average percentage of correctly pronounced words (97.68% and 97.67% respectively) that accounted for an average of 68/70 words (2 errors).

The Mann-Whitney test was used to identify differences in the intelligibility between the group of 8 Parkinsonian subjects that scored lower in the FDA (TZS2, KD4, SB5, PM7, BI8, SI10, GEI13, KA15) and the group of their matched pair controls (BA3, BE1, BI6, KP9, AN2, SB7, THM5, PE10). The results of Mann Whitney U analysis indicated no significant between group differences in intelligibility (Mann Whitney U = 23.5, Z = -0.917, $p > 0.05$). Descriptively, the Parkinsonian group had a slightly lower mean percentage of intelligibility (96.96%) than the control group (97.32%) that accounted for 2 errors for both groups.

4.3 Results in electrolaryngographic measures

The eight experimental subjects who scored lower than their controls in the FDA indicated a group of Parkinsonian subjects that may be considered as mildly dysarthric. Out of these eight subjects, as mentioned in the beginning of this chapter, six were used in the analysis of aspects of voice (BI8, GEI13, KD4, PM7, SI10 and TZS2).

The Parkinsonian mildly dysarthric and the control groups were compared in three speaking tasks: sustained phonation, reading, and conversation. The results of electrolaryngographic measures in sustained phonation are presented first and

are followed by the results of the main effect of Parkinson's disease and speaking task (reading and conversation) on voice.

Due to the small size of the sample, both descriptive analysis and statistical significance testing were employed. The descriptive analysis was used to show possible tendencies in the individual scores of the subjects, while the statistical analysis was used to examine if there are differences between the means of the groups. Statistical significance was set at 0.05 level with no Bonferroni corrections because each T-test or analysis of variance (ANOVA) was completed separately.

Logarithmically transformed data were used to ensure normality of the distributions of the groups. The use of data transformations in the analysis of the results presents some advantages. These involve the remedy of outliers and the remedy of failures of normality. Tabachnick and Fidell (2001) discuss the major advantages and disadvantages of data transformations by emphasising that an improvement in the results of the analysis occurs after transformations of almost every data set.

4.3.1 Results in sustained phonation

In review, the following variables were used in the current study in sustained phonation:

- Average fundamental frequency and standard deviation
- Average closed vocal fold contact, standard deviation and range (Qx)
- Jitter and shimmer ('short-term' cycle to cycle variations in frequency and amplitude).

Table 9 presents a summary of the electrolaryngographic results in sustained phonation including variables, descriptive data, and T-tests. Independent samples T-tests were used to identify possible differences in sustained phonation. T-tests did not find any statistically significant differences between the Parkinsonian mildly dysarthric group and its matched pair control group in any logarithmically transformed variable.

Table 9. Summary results including variables, descriptive data, and t-tests (N = 6).

VARIABLE	DESCRIPTIVE DATA Comparison of the scores between the two groups	T – tests
Average Fx (Hz)	- Decreased scores in 4 of 6 PD subjects - Increased scores in PD and controls females compared to males	p > 0.05
Standard deviation Fx (Hz)	- Increased scores in 4 of 6 PD subjects	p > 0.05
Average Qx (%)	- Decreased scores in 5 of 6 PD subjects	p > 0.05
Qx standard deviation (%)	- Equal distribution of increased and decreased scores	p > 0.05
Qx Range (%)	- Equal distribution of increased and decreased scores	p > 0.05
Jitter First (%)	- Equal distribution of increased and decreased scores	p > 0.05
Shimmer First (%)	- Decreased scores in 4 of 6 PD subjects	p > 0.05

Table 10 shows a summary of the statistics (means and standard deviations) of all variables measured during sustained phonation between the Parkinsonian mildly dysarthric group and its matched pair control group.

Table 10. Summary statistics of the variables measured during sustained phonation between Parkinsonian mildly dysarthric and control groups (N = 6).

VARIABLE	GROUP			
	Parkinson		Control	
	Mean	SD	Mean	SD
Average Fx (Hz)	170.68	66.89	153.52	29.06
Standard deviation Fx (Hz)	1.94	1.03	1.52	0.34
Average Qx (%)	40.37	3.67	46.09	6.39
Qx standard deviation (%)	0.04	0.06	0.01	0.02
Qx Range (%)	4.32	2.34	4.75	1.82
Jitter First (%)	0.99	1.25	0.48	0.24
Shimmer First (%)	4.05	1.86	4.64	2.46

An examination of the individual scores of the two groups showed no specific patterns according to age and gender. Only in the variable average Fx, both the Parkinsonian and control female scores were higher than male scores. Moreover, the scores of the two female Parkinsonian subjects (TZS2, GEI13) compared to their controls (BA3, THM5) showed in all variables opposite tendencies. This may be due either to age difference (20 years age difference between the two female Parkinsonian subjects) or idiosyncratic differences. Figure 26 shows the matched pair scores in the average Fx variable.

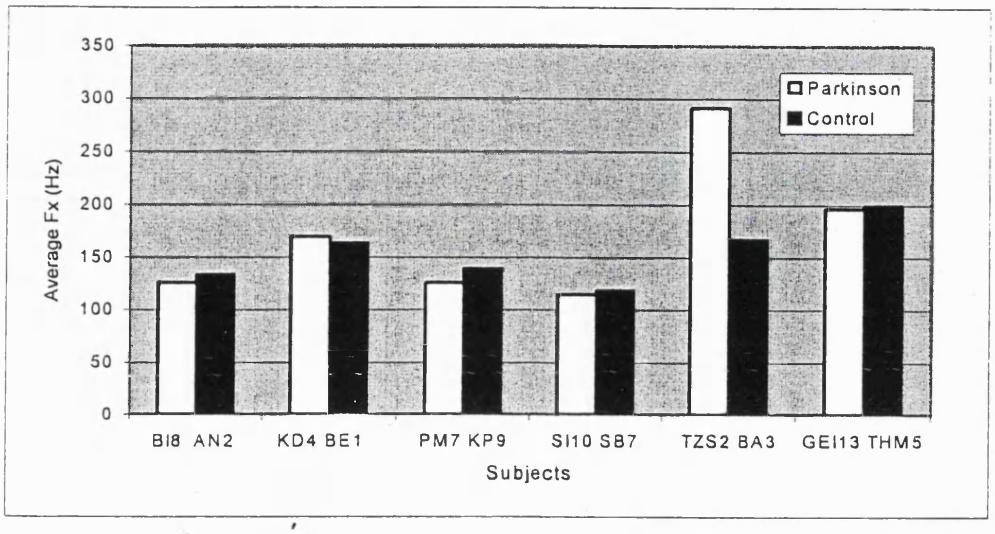

Figure 26. Matched pair scores in the average Fx variable

The variable average Qx was the only one where the pattern of decreased individual scores of the Parkinsonian mildly dysarthric group compared to its control group is in accordance with the group means. Figure 27, shows the matched pair scores in average Qx variable.

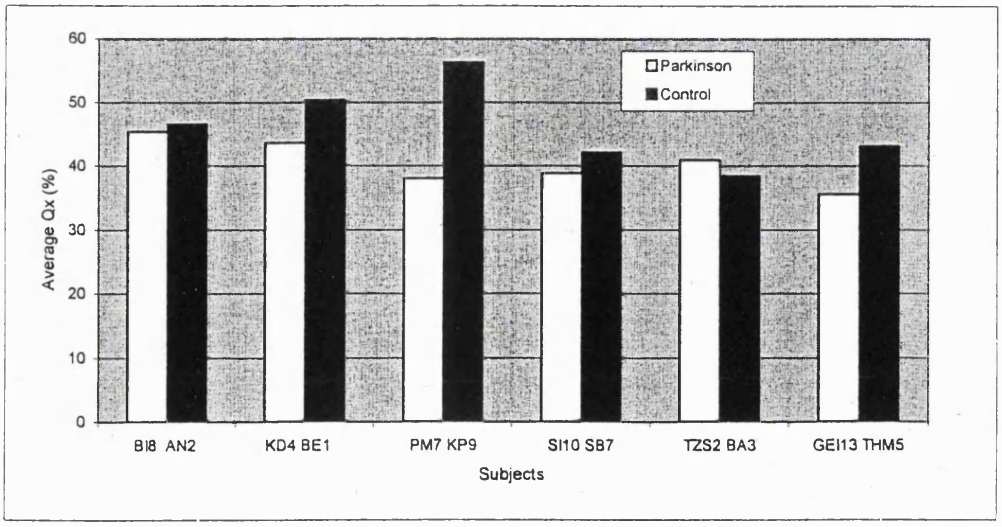

Figure 27. Matched pair scores in average Qx variable.

4.3.2 Results of the main effect of Parkinson's disease and speaking task (reading and conversation) on voice

In review, the following variables were used in the current study in reading and conversation:

- Mean fundamental frequency, standard deviation and range (DFx1 and DFx2)
- Mean relative intensity, standard deviation and range (DAx1 and DAx2)
- Mean closed vocal fold contact, standard deviation and range (DQx1).

Table 11 presents a summary of the electrolaryngographic results in reading and conversation including variables, descriptive data, and analysis of variance (ANOVA). The data were analyzed using a two-way analysis of variance in which one variable of interest was Parkinson's disease and the other was speaking task to observe their effects on aspects of voice. The results did not show any statistically significant differences between the Parkinsonian mildly dysarthric group and its matched control group in any logarithmically transformed variable.

Table 11. Summary results including variables, descriptive data and anovas (N = 6)

VARIABLE – SPEAKING TASK	DESCRIPTIVE DATA Comparison of scores between the two groups	ANOVA
DFx1 Mean in Conv. (Hz)	- Increased scores in 5 of 6 PD subjects - Increased scores in PD and controls females compared to males	p > 0.05
DFx1 Mean in Read. (Hz)	- No pattern - Increased scores in PD and controls females compared to males	p > 0.05
DFx1 SD in Conv. (Hz)	- No pattern	p > 0.05
DFx1 SD in Read. (Hz)	- No pattern	p > 0.05
DFx1 90% Range in Conv. (Octave)	- Equal distribution of increased and decreased scores	p > 0.05
DFx1 90% Range – Read. (Octave)	- Decreased scores in 4 of 6 PD subjects	p > 0.05
DFx2 Mean – Conv. (Hz)	- Increased scores in 5 of 6 PD subjects - Increased scores in PD and controls females compared to males	p > 0.05
DFx2 Mean – Read. (Hz)	- No pattern - Increased scores in PD and controls females compared to males	p > 0.05
DFx2 SD – Conv. (Hz)	- No pattern	p > 0.05
DFx2 SD – Read. (Hz)	- No pattern	p > 0.05
DFx2 90% Range – Conv. (Octave)	- Equal distribution of increased and decreased scores	p > 0.05
DFx2 90% Range – Read. (Octave)	- Decreased scores in 4 of 6 PD subjects	p > 0.05
DAx1 Mean – Conv. (dB)	- Decreased scores in 5 of 6 PD subjects	p > 0.05
DAx1 Mean – Read. (dB)	- No pattern	p > 0.05
DAx1 SD – Conv. (dB)	- No pattern	p > 0.05
DAx1 SD – Read. (dB)	- No pattern	p > 0.05
DAx1 90% Range – Conv. (dB)	- Decreased scores in 4 of 6 PD subjects	p > 0.05
DAx1 90% Range – Read. (dB)	- Equal distribution of increased and decreased scores	p > 0.05
DAx2 Mean – Conv. (dB)	- No pattern	p > 0.05
DAx2 Mean – Read. (dB)	- No pattern	p > 0.05
DAx2 SD – Conv. (dB)	- No pattern	p > 0.05
DAx2 SD – Read. (dB)	- No pattern	p > 0.05
DAx2 90% Range – Conv. (dB)	- Decreased scores in 4 of 6 PD subjects	p > 0.05
DAx2 90% Range – Read. (dB)	- Equal distribution of increased and decreased scores	p > 0.05
DQx1 Mean – Conv. (%)	- Decreased scores in 4 of 6 PD subjects	p > 0.05
DQx1 Mean – Read. (%)	- Decreased scores in 5 of 6 PD subjects	p > 0.05
DQx1 SD – Conv. (%)	- No pattern	p > 0.05
DQx1 SD – Read. (%)	- No pattern	p > 0.05
DQx1 90% Range – Conv .(%)	- Decreased scores in 4 of 6 PD subjects - Decreased scores of Parkinsonian females compared to their controls and to the males	p > 0.05
DQx1 90% Range – Read. (%)	- Equal distribution of increased and decreased scores - Decreased scores of Parkinsonian females compared to their controls and to the males	p > 0.05

Table 12 below shows summary statistics of the variables measured during reading and conversation (means and standard deviations) in the two groups.

Table 12. Summary statistics of the variables measured during reading and conversation between Parkinsonian mildly dysarthric and the control groups (N = 6).

VARIABLE – SPEAKING TASK	STUDY GROUP			
	Parkinson		Control	
	Mean	SD	Mean	SD
DFx1 Mean – Conversation (Hz)	167.38	59.74	148.42	33.07
DFx1 Mean – Reading (Hz)	159.05	67.06	147.79	33.55
DFx1 Standard Deviation – Conversation (Hz)	0.12	0.03	0.12	0.02
DFx1 Standard Deviation – Reading (Hz)	0.08	0.01	0.08	0.02
DFx1 90% Range – Conversation (Octave)	1.15	0.39	1.21	0.33
DFx1 90% Range – Reading (Octave)	0.62	0.14	0.77	0.25
DFx2 Mean – Conversation (Hz)	174.46	56.95	154.59	36.40
DFx2 Mean – Reading (Hz)	163.72	69.04	152.35	31.67
DFx2 Standard Deviation – Conversation (Hz)	0.05	0.09	0.06	0.02
DFx2 Standard Deviation – Reading (Hz)	0.03	0.006	0.04	0.009
DFx2 90% Range – Conversation (Octave)	0.93	0.27	0.89	0.18
DFx2 90% Range – Reading (Octave)	0.50	0.13	0.61	0.15
DAx1 Mean – Conversation (dB)	56.11	6.11	54.81	7.40
DAx1 Mean – Reading (dB)	58.05	6.17	57.04	6.88
DAx1 Standard Deviation – Conversation (dB)	8.72	1.31	8.52	1.79
DAx1 Standard Deviation – Reading (dB)	7.70	1.42	7.94	1.50
DAx1 90% Range – Conversation (dB)	16.10	2.65	15.88	3.14
DAx1 90% Range – Reading (dB)	14.08	2.76	14.77	2.88
DAx2 Mean – Conversation (dB)	57.22	6.25	56.02	7.69
DAx2 Mean – Reading (dB)	59.07	6.26	58.42	6.42
DAx2 Standard Deviation – Conversation (dB)	3.67	0.34	3.60	0.74
DAx2 Standard Deviation – Reading (dB)	3.19	0.30	3.32	0.45
DAx2 90% Range – Conversation (dB)	15.18	2.78	14.65	2.91
DAx2 90% Range – Reading (dB)	12.12	2.29	13.02	3.10
DQx1 Mean – Conversation (%)	38.50	5.80	42.33	4.96
DQx1 Mean – Reading (%)	38.33	5.85	43.83	6.35
DQx1 Standard Deviation – Conversation(%)	5.82	1.51	7.31	1.34
DQx1 Standard Deviation – Reading (%)	4.80	0.91	5.76	1.40
DQx1 90% Range – Conversation (%)	17.35	5.10	22.45	4.94
DQx1 90% Range – Reading (%)	13.80	3.11	17.55	4.54

Careful observation of the individual scores in fundamental frequency (DFx1 mean and DFx2 mean) in the conversation task showed a pattern of increased individual scores of the Parkinsonian mildly dysarthric subjects compared to their matched pairs. This pattern is in accordance with the group means of these variables. One subject (PM7) showed a lower score as compared to its matched pair control. However, this subject had been exposed to levodopa medication 6 months before the neurological diagnosis and this may account for that finding. Higher fundamental frequency in Parkinson's disease has been associated with the rigidity of the vocal folds (Hanson et al., 1983). However, this trend was not the same in reading where 2 Parkinsonian mildly dysarthric subjects showed higher scores, 2 showed lower scores and the last 2 subjects showed equal scores with their matched pair controls. Moreover, gender differences were observed in the DFx1 and DFx2 mean variables in both reading and conversation tasks. Female scores (TZS2 and GEI13) were increased compared to male scores (in both groups). Even though no normative data in fundamental frequency according to gender exist in the Greek language, this pattern of increased female scores is in accordance with English studies (Baken, 1997; Greene & Mathieson, 1997; Linville, 2000). Figures 28-31 show the matched pair scores in DFx1 and DFx2 mean variables.

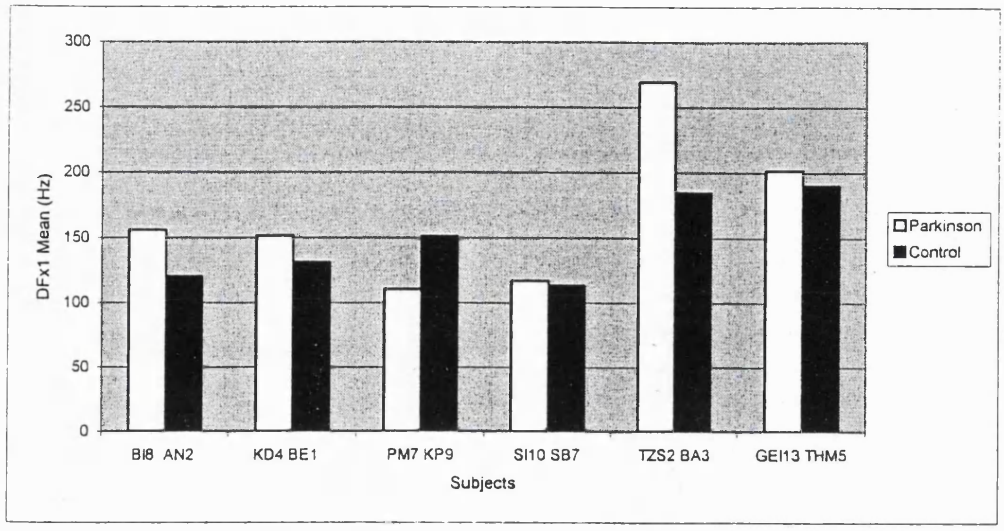

Figure 28. Matched pair scores in DFx1 mean variable in conversation.

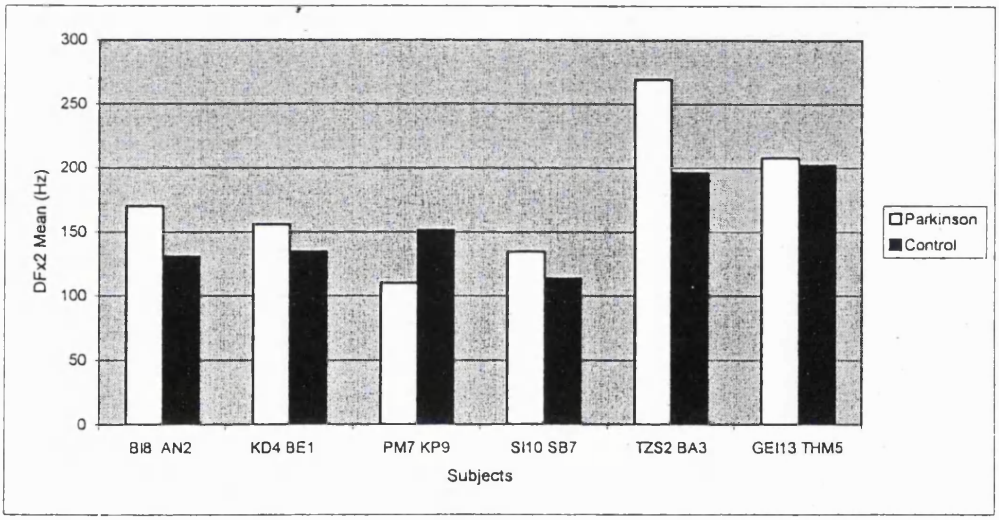

Figure 29. Matched pair scores in DFx2 mean variable in conversation.

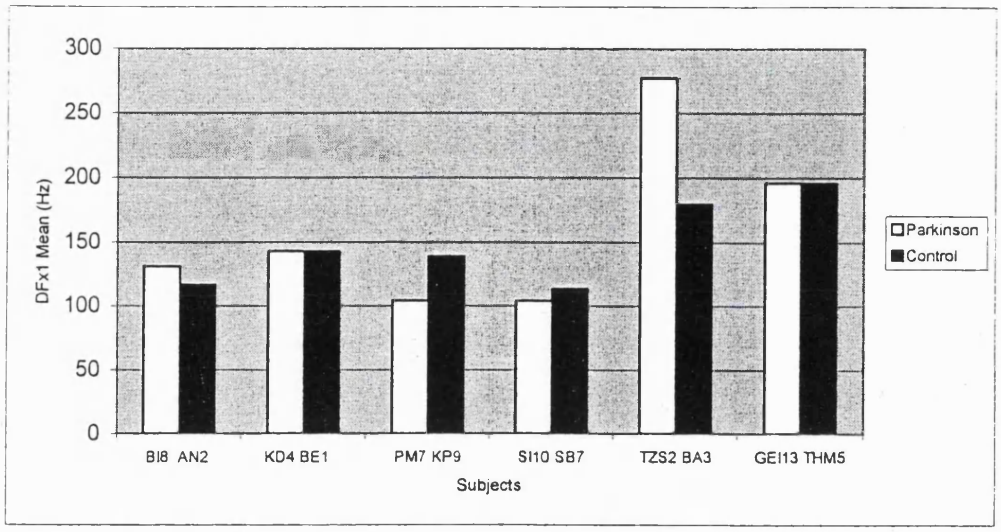

Figure 30. Matched pair scores in DFx1 mean variable in reading.

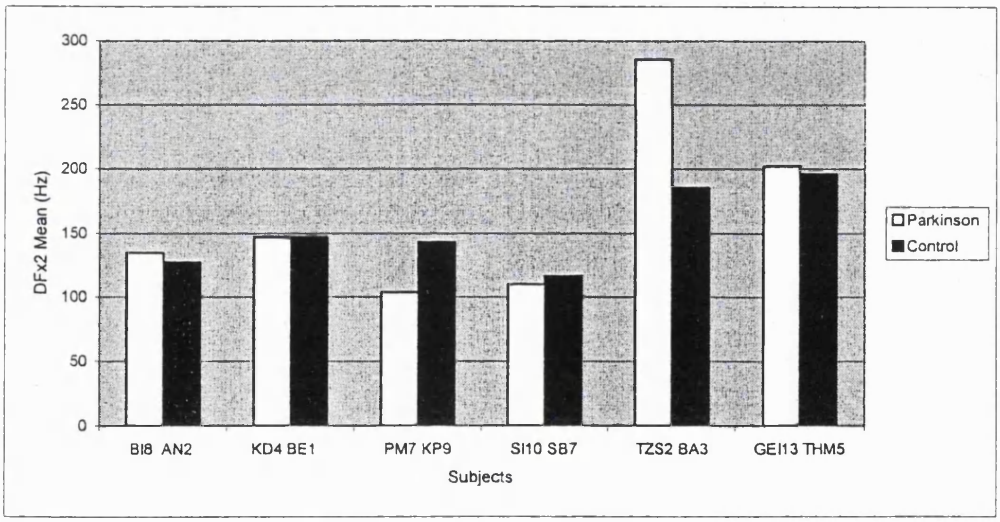

Figure 31. Matched pair scores in DFx2 mean variable in reading.

In relative intensity, a pattern of decreased scores in the Parkinsonian mildly dysarthric subjects was observed in DAx1 mean variable in conversation and not in reading. However, this pattern did not reappear in DAx2 mean in neither speaking tasks. Since, DAx2 mean is analogous to DFx2 mean which measures vocal fold vibrational regularity (Fourcin, 2000) the aforementioned pattern can be considered

as accidental. In the range of relative intensity (DAx1 90% mean range and DAx2 90% mean range) a tendency of decreased scores in Parkinsonian mildly dysarthric subjects in conversation was found. This tendency is not in accordance with the group means. No pattern was found in reading.

In voice quality quotients (DQx1 mean) a pattern of decreased scores in the Parkinsonian mildly dysarthric subjects was observed in both speaking tasks. This pattern is in accordance with the group means. As already has been said, the same pattern was observed in average Qx mean variable in sustained phonation. Appendix P shows the aforementioned tendencies of the individual scores in the variables mean DFx1 and mean DFx2 in conversation, mean DQx1 in reading and conversation, and average Qx in sustained phonation. Some authors state that Qx as a ratio of closed vocal fold contact duration to the total period is related to the quality of phonation (Abberton & Fourcin, 1984; Fourcin, 2000). The longer closed phase or a bigger Qx ratio might indicate a 'better' voice. Consequently, the tendencies in the mean Qx variables that were found lower in the Parkinsonian mildly dysarthric group might indicate a tendency for a breathier quality of phonation for that group as compared to the control group. Physiologically, this might indicate a proportionally increased open phase as compared to the closed phase of the vocal folds. Figure 32 and 33 show the matched pair scores in DQx1 mean variable in conversation and reading.

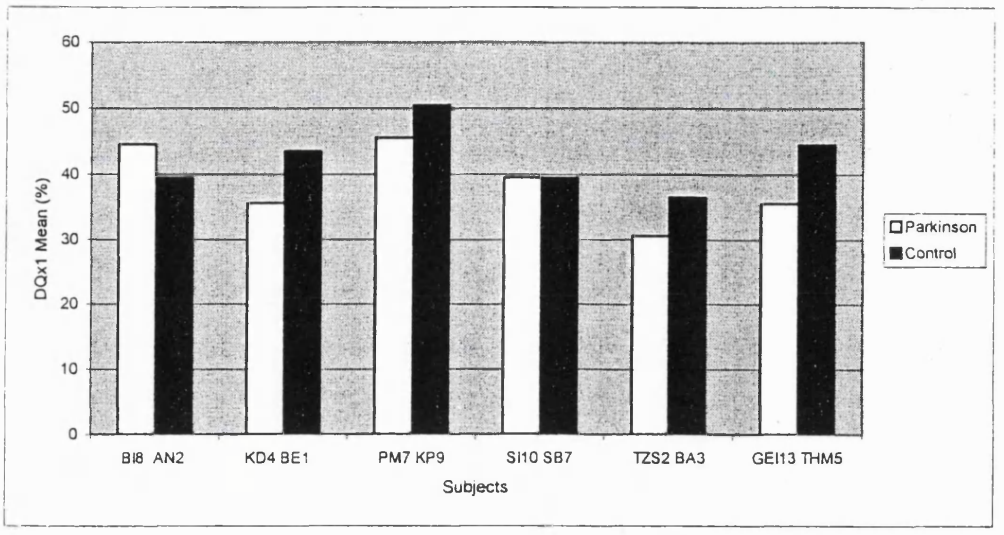

Figure 32. Matched pair scores in DQx1 mean variable in conversation.

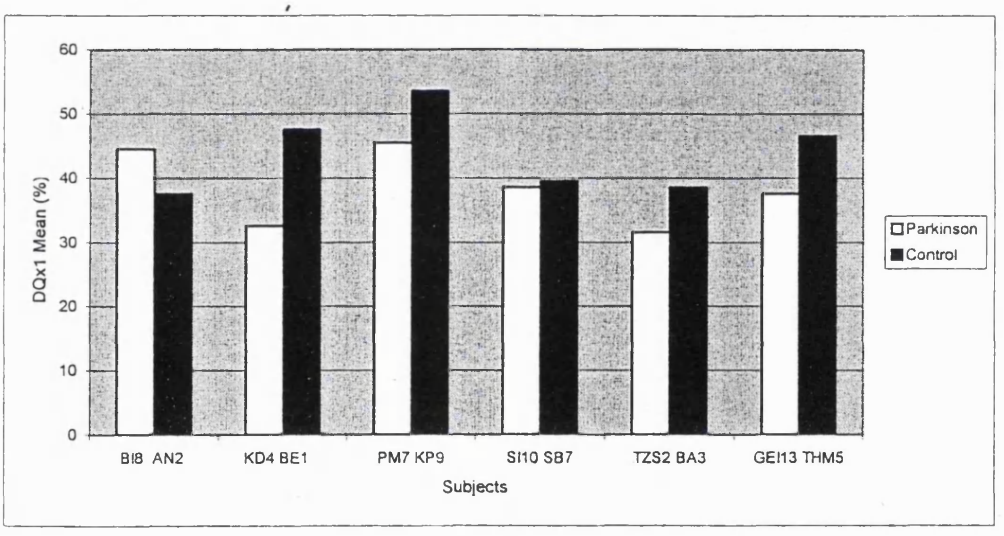

Figure 33. Matched pair scores in DQx1 mean variable in reading.

Finally, gender differences were observed in the DQx1 90% mean range variable in both reading and conversation tasks. Scores of the female Parkinsonian subjects were decreased compared to their matched pairs and the males.

In the lack of no statistically significant differences between the means of the group variables, the above observations could be explained only as tendencies that

may become clearer as the disease progresses. Further research is needed to explore this assumption.

The observation of the individual scores in reading compared to conversation showed discrepancies between these tasks. Only, in DQx1 mean variable there was the same tendency of decreased scores in Parkinsonian mildly dysarthric subjects across speaking tasks. In all other variables, observation of raw data showed differences across speaking tasks. Even though, repeated measures analysis of variance (ANOVA) was applied to identify if there was a main effect of speaking task (reading as compared to conversation) on voice, its results should be taken with caution since the individual scores did not show the same tendencies with the means. In the Parkinsonian mildly dysarthric group and its matched control group (N = 6) statistically significant differences were found in the following variables:

- Standard deviation DFx1 (SDDFx1)
- DFx1 90% range
- Standard deviation DFx2 (SDDFx2)
- DFx2 90% range
- DAx1 mean
- DAx1 90% range
- DAx2 mean
- Standard deviation DAx2 (SDDAx2)
- DAx2 90% range
- Standard deviation DQx1 (SDDQx1)
- DQx1 90% range.

Table 13 below shows summary statistics of the Parkinsonian mildly dysarthric and the control groups in the variables that were found to be significant in the main effect of speaking task on voice. Appendix O shows the results in reading as compared to conversation in all variables between the groups.

Table 13. Summary statistics of the variables that were found to be significant in the main effect of speaking task on voice (N = 6).

		Parkinson's Group				Control Group			
		Mean	SD	Median	Range	Mean	SD	Median	Range
Standard Deviation DFx1 (Hz)	Conversation	0.12	0.03	0.12	0.08	0.12	0.02	0.12	0.05
	Reading	0.08	0.01	0.08	0.04	0.09	0.02	0.08	0.07
DFx1 90% Range (Hz)	Conversation	1.15	0.39	1.13	0.88	1.21	0.33	1.14	0.76
	Reading	0.62	0.14	0.62	0.40	0.77	0.25	0.67	0.66
Standard Deviation DFx2 (Hz)	Conversation	0.05	0.009	0.05	0.02	0.06	0.02	0.06	0.05
	Reading	0.03	0.006	0.03	0.02	0.04	0.009	0.04	0.02
DFx2 90% Range (Hz)	Conversation	0.93	0.27	0.84	0.72	0.90	0.18	0.93	0.43
	Reading	0.50	0.13	0.50	0.35	0.61	0.15	0.59	0.40
DAx1 Mean (dB)	Conversation	56.11	6.11	54.72	17.78	54.81	7.40	54.17	22.22
	Reading	58.06	6.17	56.67	17.22	57.04	6.88	55.28	20.00
DAx1 90% Range (dB)	Conversation	16.10	2.65	16.70	6.10	15.88	3.14	16.00	9.20
	Reading	14.08	2.76	13.85	7.40	14.77	2.88	14.05	7.50
DAx2 Mean (dB)	Conversation	57.22	6.25	56.11	18.33	56.02	7.69	55.83	23.34
	Reading	59.07	6.26	58.06	17.78	58.43	6.42	56.95	18.89
Standard Deviation DAx2 (dB)	Conversation	3.67	0.34	3.76	0.97	3.60	0.74	3.56	2.20
	Reading	3.19	0.30	3.20	0.79	3.32	0.45	3.32	1.34
DAx2 90% Range (dB)	Conversation	15.18	2.78	15.40	6.80	14.65	2.91	14.20	7.80
	Reading	12.12	2.29	12.40	6.80	13.02	3.10	11.90	8.00
Standard Deviation DQx1 (%)	Conversation	5.82	1.51	6.14	3.41	7.32	1.34	7.15	3.61
	Reading	4.80	0.91	4.81	2.75	5.77	1.40	5.33	3.64
DQx1 90% Range (%)	Conversation	17.35	5.10	17.40	11.10	22.45	4.94	20.80	11.40
	Reading	13.80	3.11	13.85	8.40	17.55	4.54	16.35	11.70

The lack of differences in mean fundamental frequency (DFx1 and DFx2) across speaking tasks (reading and conversation) in both groups support the observations by Brown and Docherty (1995) who found no changes in mean fundamental frequency across speaking tasks in a dysarthric and a control group.

In general, differentiation across speaking tasks is a characteristic shown most notably in normal rather than dysarthric speakers (Leuschel & Docherty, 1996, 2001). The results of the present study of the main effect of speaking task on voice do not support the findings by other researchers (Leuschel & Docherty, 1996, 2001) who showed no differentiation of speaking task in dysarthric speakers. No accordance of individual scores to the means (present study) is a prohibiting factor to the explanation of these results. Also, different instrumentation, differential selection of subjects and different severity level of dysarthria may be responsible for these discrepancies in the Leuschel and Docherty study. For example, only half of their subjects in both studies included Parkinsonian patients while the remaining included subjects with Multiple Sclerosis. The above observations support the notion that both tasks should be involved not only in speech (prosodic) assessment of dysarthria (Brown & Docherty, 1995; Leuschel & Docherty, 1996, 2001) but also in voice assessment (Fox & Ramig, 1997).

4.4 Summary of the between group results

In summary, 8 out of 12 subjects who were diagnosed with early Parkinson's disease exhibited lower scoring in the Frenchay Dysarthria Assessment (FDA) as compared to a group of matched pair controls. This lower scoring involved primarily the tongue area (decreased in speed movement of the tongue) and followed by the

laryngeal area (voicing time in extended sustained phonation, volume and pitch) and finally the lips area (decreased in speed movement of the lips).

No statistically significant intelligibility differences were found when either the entire Parkinsonian group or the Parkinsonian mildly dysarthric group who exhibited lower scoring in the FDA were compared to a matched pair control group. However, observation of the individual scores in the Parkinsonian mildly dysarthric group and its matched pair control group showed a tendency for slightly lower scores in intelligibility for the first group.

Electrolaryngographic measures were used to quantify aspects of voice in the two groups. The effect of Parkinson's disease and the effect of speaking task on voice were measured. No statistically significant differences were found between the Parkinsonian mildly dysarthric subjects and their matched pair controls in the effect of Parkinson's disease on voice in all speaking tasks. Observation of the individual scores showed a tendency of the Parkinsonian mildly dysarthric group for higher scores in fundamental frequency (DFx1 and DFx2) in conversation and for lower scores in voice quality (average Qx and DQx1) in all speaking tasks. The tendencies of DFx1 and DFx2 scores in conversation might be precursors of the stiffening of the vocal folds (rigidity) while the tendency in Qx scores in all speaking tasks might be a precursor of a breathier voice quality. Both, observation of individual scores and statistical analyses showed differentiation across speaking tasks (reading compared to conversation). The findings of the differentiation of reading as compared to conversation in the majority of voicing variables prove the overlapping of both groups and the normality in their scores. Dysarthria in the beginning of Parkinson's disease appears to exhibit itself in isolated movements of the articulators rather than in phonatory measures.

CHAPTER 5. RESULTS WITHIN GROUP (MEDICATION)

This chapter describes the effects of medication on speech and voice. The structure of the results, as in chapter 4, follows the order of the speech assessment:

- The results of the Frenchay Dysarthria Assessment (FDA)
- The results of the intelligibility testing
- The electrolaryngographic (ELG) measurements.

In all of the above measurements, the Parkinsonian subjects who scored lower in the FDA (mildly dysarthric group), were used to observe the effect of medication (BI8, GEI13, KD4, PM7, SI10, TZS2). As mentioned in chapter 4, one subject (KA15), in whom it was not possible to establish ELG signal, left the study after beginning medication. Another subject (SB5) stopped taking medication two months after the initiation of therapy. Therefore, he was treated as a separate case and his results in aspects of voice are compared against the Parkinsonian mildly dysarthric group of males.

Subject PX9 was not included in the between group results because she reported laryngeal problems due to surgery of the parathyroid and one thyroid gland 10 years before the diagnosis of Parkinson's disease. This subject has never received voice therapy. PX9 was included in the within group results, to observe the effect of medication on the Frenchay Dysarthria Assessment (FDA), and especially on its maximum performance subtests (movement of tongue and lips). It was decided to include her also in the intelligibility and ELG analyses since she was

diagnosed with Parkinson's disease. However, the analyses took place both with and without her to avoid her laryngeal pathology confound the results.

5.1 Results of the Frenchay Dysarthria Assessment

The Frenchay Dysarthria Assessment (FDA) was re-administered to mildly dysarthric Parkinsonian subjects 3-3.5 months after starting medication. The results of the FDA are shown below in table 14. The positive sign (+) shows the areas in which the medication had a positive effect (i.e., b to a), the negative sign (-) shows the areas in which the medication had a negative effect (i.e., a to b) and the equal sign (=) shows the areas in which there was no effect. Table 14 also includes the remaining subjects of the experimental group (LI14, GN11, KP6), whose FDA results before medication showed no signs of dysarthria ("a" score), in order to examine if the medication had a negative effect on their speech. The findings showed no difference in the non dysarthric Parkinsonian subjects. In general, palate was an area of the FDA where no differences were found while reflex was an area in which two subjects gave different outcomes. PM7 had a positive outcome (+) while PX9 showed a negative outcome (-). Because reflex alone does not contribute much in dysarthria and because the majority of research in dysarthria (perceptual, acoustic and physiological) focus on the areas of lips, laryngeal and/or tongue, the reflex area was omitted from the table 14 (even though it was included in individual figures).

Table 14. Differences of the Parkinsonian group before and after medication in the Frenchay Dysarthria Assessment.

Subjects	Lips	Laryngeal	Tongue
Males			
KD4 before vs. KD4 after	=	+	+
KP6 before vs. KP6 after	*No difference*		
PM7 before vs. PM7 after	=	-	-
BI8 before vs. BI8 after	+	=	+
SI10 before vs. SI10 after	No difference		
GN11 before vs. GN11 after	*No difference*		
LI14 before vs. LI14 after	*No difference*		
Females			
TZS2 before vs. TZS2 after	+	+	+
GEI13 before vs. GEI13 after	No difference		
PX9 before vs. PX9 after	+	-	+

Note: The three subjects presented in italics are the non dysarthric subjects.

Two mildly dysarthric subjects (GEI13, SI10) out of seven exhibited no differences in their FDA results before and after medication. Five subjects were found to exhibit differences in the Frenchay Dysarthria Assessment (FDA) before medication as compared to after medication condition (KD4, PM7, BI8, TZS2 and PX9). The outcome of medication in every subject was defined as the total number of his/her positive (+), negative (-) or equal signs (=). Four out of these five subjects (KD4, BI8, TZS2 and PX9) exhibited an overall individual positive outcome in the areas of lips, laryngeal and tongue (more positive signs than negative signs). One subject (PM7) exhibited an overall negative outcome (two negative signs).

In general, the tongue area of the FDA was the area where the medication had an overall positive impact across the mildly dysarthric group (4 positive signs and 1 negative sign), followed by the lips (3 positive signs) and the laryngeal area (2

positive signs and 2 negative signs). The results of the five Parkinsonian mildly dysarthric subjects where differences were found are presented below in figures 34-38, in a descending order of the positive outcome of medication (subjects who exhibited more in number positive signs are presented first). The subsections of the Frenchay dysarthria assessment that are presented in figures 34-38 involve differences of a least a scale (i.e. a to b) in each subject before and after medication. All the other subsections did not present differences before and after medication.

TZS2 was the subject in whom the medication had the most impact in the area of lips and less impact in the areas of laryngeal and tongue. Figure 34 below shows the areas of the FDA that TZS2 differed in before and after medication condition.

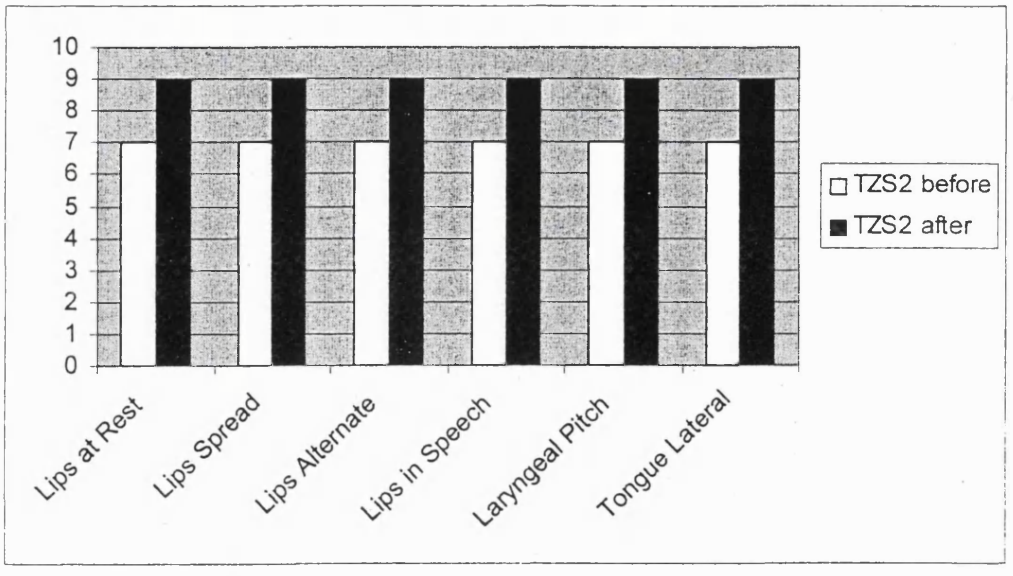

Figure 34. Results of the Frenchay Dysarthria Assessment before and after medication for subject TZS2.

BI8 was the subject in whom the medication had the most impact in the area of tongue and less impact in the area of lips. Figure 35 below shows the areas of the FDA that BI8 differed in before and after medication.

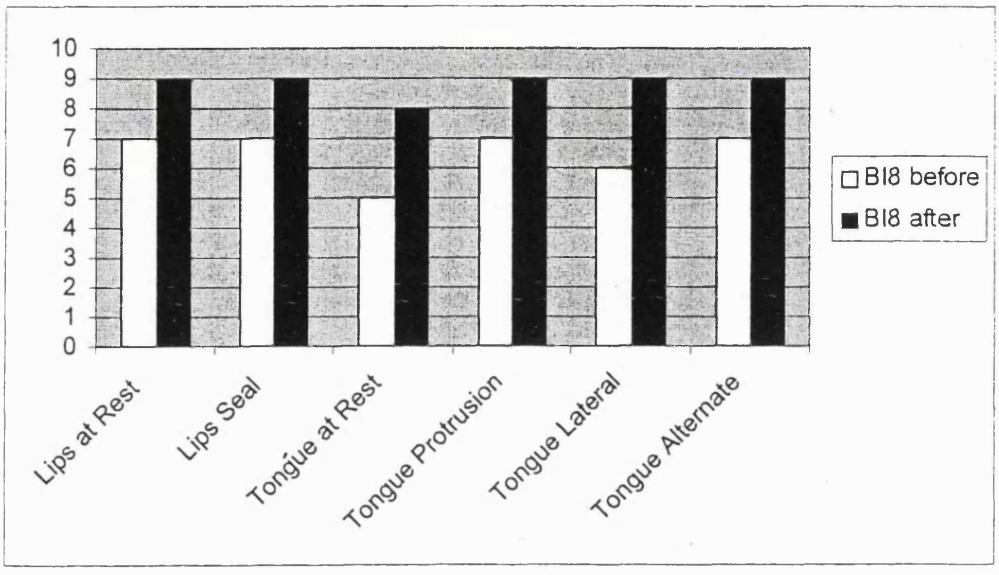

Figure 35. Results of the Frenchay Dysarthria Assessment before and after medication for subject BI8.

KD4 was the subject in whom the medication had the most impact in tongue and less impact in the laryngeal area (Figure 36).

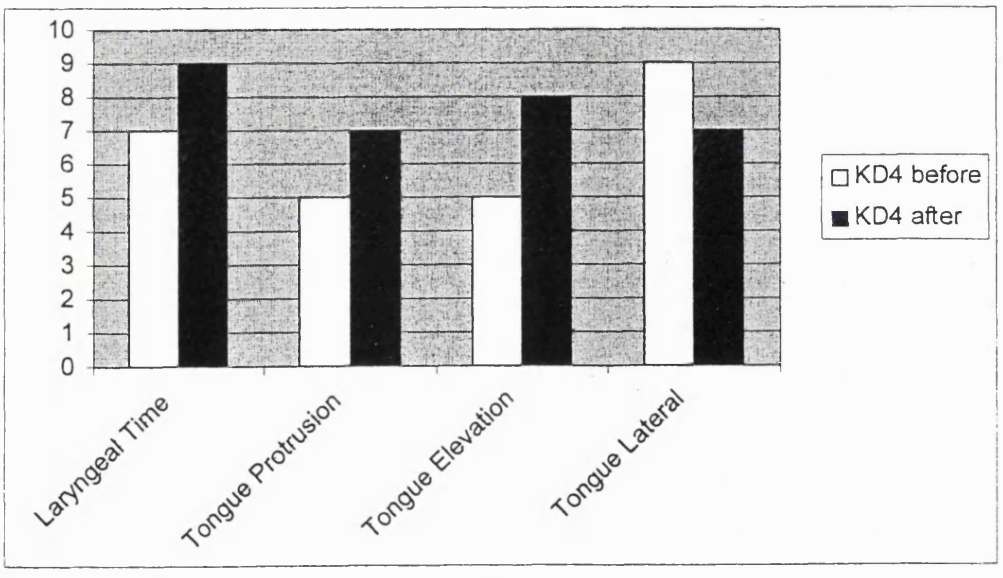

Figure 36. Results of the Frenchay Dysarthria Assessment before and after medication for subject KD4.

In subject PX9, medication had the most positive impact in the areas of tongue and lips and the most negative impact in the areas of laryngeal and reflex (Figure 37). This subject reported laryngeal pathology due to surgery in 1990. In this study, her voice was perceived as harsh both before and after levodopa medication. During the completion of the history form before medication, PX9 perceived her speech to be slower in the past 2 years. In the post medication session she reported better movement of her limbs and better speech and voice. Her scores in the laryngeal and reflex sections however, were lower after medication (more time to complete the task). The discrepancy of her scores in the FDA with her perception might be due to the fact that the laryngeal time and pitch tasks are tasks that aim to bring the larynx and tongue to their extremes (maximum performance tests) and they are not used in a natural conversation. Sustaining an [a] for as long as possible (laryngeal time) is not a task that people use in their voice every day. However, it seems interesting that the improvement of the performance of PX9 in the lips and tongue areas follows the trend of the majority of subjects.

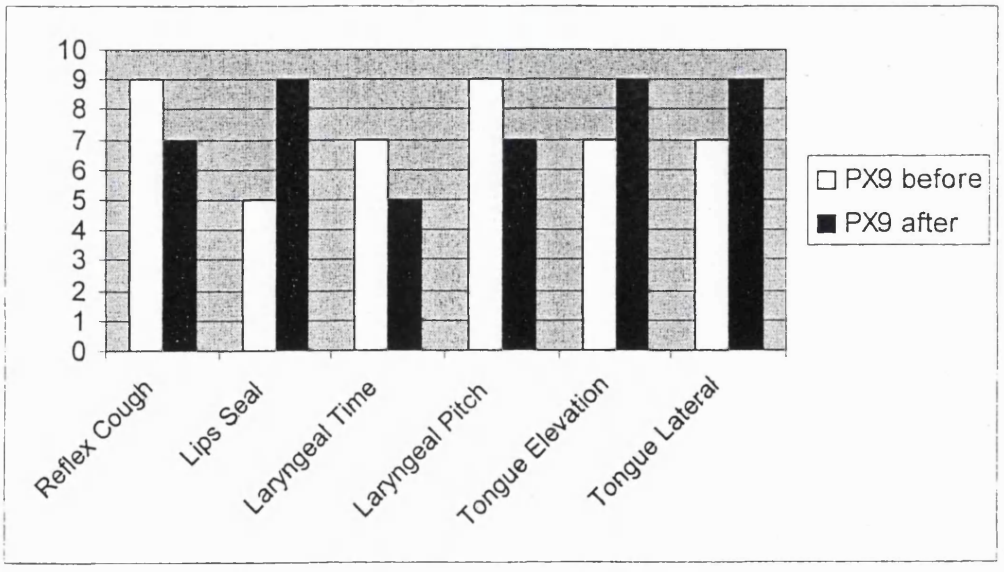

Figure 37. Results of the Frenchay Dysarthria Assessment before and after medication for subject PX9.

PM7 was the only subject in whom the medication had a negative impact in all areas of reflex, laryngeal and tongue (Figure 38). PM7 had received a number of medications that he reported to have stopped 6 months before the beginning of this study. This medication involved drugs such as Akineton (12/6/1998-17/12/1998), Sinemet (24/12/1998-6/1999) and Symmetrel (26/1/1999-6/1999). Even though he reported to have stopped medication 6 months before he was diagnosed with Parkinson's disease from the consultant neurologist in the university clinic, the influence of different drugs and/or the progression of the disease itself, probably had a negative effect on his speech/voice.

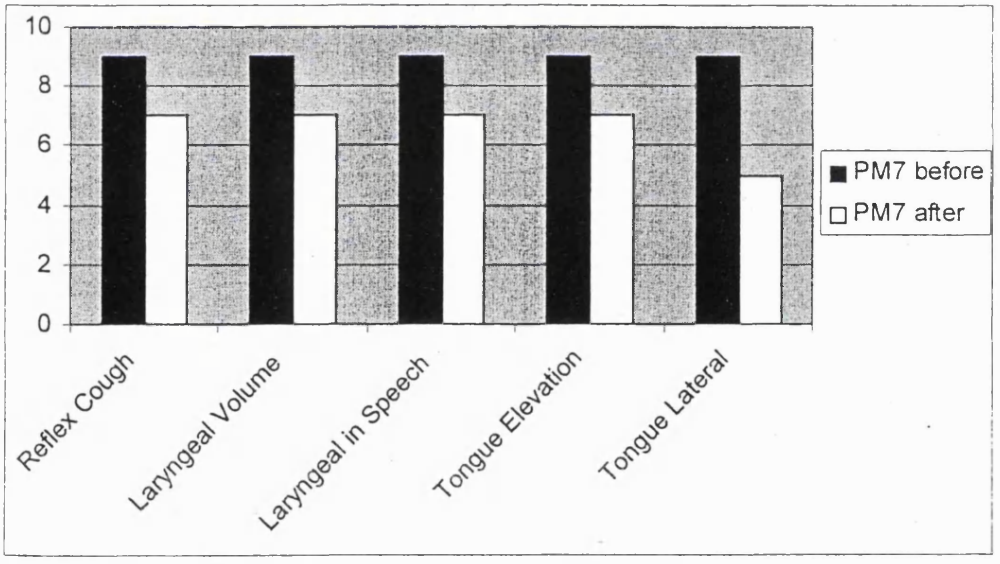

Figure 38. Results of the Frenchay Dysarthria Assessment before and after medication for subject PM7.

5.2 Intelligibility results

The intelligibility scores of the seven Parkinsonian mildly dysarthric subjects before and after medication are shown below in table 15. All intelligibility scores are in the form of a percentage and an average number of errors. Careful observation of the scores shows that all mildly dysarthric subjects exhibited an slight increase in intelligibility after medication. An exception to this finding was the subject PX9 (with the laryngeal pathology) in whom the intelligibility percentage lowered and the subject PM7 in whom the intelligibility percentage stayed the same.

Table 15. Intelligibility scores of the Parkinsonian group before and after medication.

Experimental Subjects Before Medication	Intelligibility Scoring	Experimental Subjects After Medication	Intelligibility Scoring
TZS2	96.42% (67.5/70)	TZS2	97.85% (68.5/70)
KD4	98.57% (69/70)	KD4	99.28% (69.5/70)
PM7	95.00% (66.5/70)	PM7	95.00% (66.5/70)
BI8	98.57% (69/70)	BI8	99.28% (69.5/70)
SI10	96.42% (67.5/70)	SI10	98.57% (69/70)
GEI13	97.14% (68/70)	GEI13	99.28% (69.5/70)
PX9	98.57% (69/70)	PX9	97.14% (68/70)
Group Mean	97.24%	Group Mean	98.06%
Standard Deviation	1.40	Standard Deviation	1.58
Group Mean Excluding Subject PX9	97.02%	Group Mean Excluding Subject PX9	98.21%
Standard Deviation	1.39	Standard Deviation	1.67

Statistical analyses to identify possible group differences took place. The Wilcoxon paired samples test was used to identify differences in the means of the Parkinsonian mildly dysarthric group before and after medication. The results showed that no statistically significant differences in intelligibility were found $t(6) = -1.702$, $p > 0.05$. Inspection of the means showed that the Parkinsonian mildly dysarthric group exhibited a higher intelligibility mean after medication.

The Wilcoxon paired samples test was used to identify differences between the Parkinsonian mildly dysarthric group before and after medication excluding the subject PX9, who except Parkinson's disease, exhibited also laryngeal pathology from another cause. Statistically significant differences in intelligibility were found $z = -2.032$, $p < 0.05$.

5.3 Results in electrolaryngographic measures

The Parkinsonian mildly dysarthric group before and after medication was compared in three speaking tasks: sustained phonation, reading, and conversation. The results of electrolaryngographic measures in sustained phonation are presented first and are followed by the results of the main effect of medication and speaking task (reading and conversation) on voice.

Due to the small size of the sample, both descriptive analysis and statistical significance testing were employed. The descriptive analysis was used to show possible tendencies in the individual scores of the subjects, while the statistical analysis was used to examine if there are differences between the means of the groups. Logarithmically transformed data were used to ensure normality in the distributions of the group before and after medication.

5.3.1 Results in sustained phonation

In review, the following variables were used in sustained phonation to compare the mildly dysarthric group before and after medication:

- Average fundamental frequency and standard deviation
- Average closed vocal fold contact, standard deviation and range (Qx)
- Jitter and shimmer ('short-term' cycle to cycle variations in frequency and amplitude).

Table 16 presents a summary of the electrolaryngographic results in sustained phonation including variables, descriptive data, and t-tests. Paired samples T-tests were used to identify possible differences. They did not find any statistically significant differences in the Parkinsonian mildly dysarthric group (N = 7) before and after medication in any logarithmically transformed variable. Special attention will be given to the individual scores of subject PX9 with the laryngeal pathology which present differences from the rest of the group and particularly of the female group.

Table 16. Summary results including variables, descriptive data, and t-tests (N = 7).

VARIABLE	DESCRIPTIVE DATA Comparison of the scores in the Parkinsonian group before and after medication	T – tests
Average Fx (Hz)	- No pattern - Increased pre and post medication scores in PD females compared to males - A large increase in PX9's score after medication compared to all subjects approaching other females scores	$p > 0.05$
Standard deviation Fx (Hz)	- No pattern	$p > 0.05$
Average Qx (%)	- Small increases in scores in 5 of 7 PD subjects after medication	$p > 0.05$

Qx standard deviation (%)	- No pattern	p > 0.05
Qx Range (%)	- No pattern	p > 0.05
Jitter First (%)	- No pattern	p > 0.05
Shimmer First (%)	- No pattern	p > 0.05

Table 17 below, shows a summary of the statistics (means and standard deviations) in all variables measured during sustained phonation.

Table 17. Summary statistics of the variables measured during sustained phonation in the Parkinsonian group before and after medication (N = 7).

VARIABLE – CONDITION	Mean	SD
Average Fx Before Medication (Hz)	168.60	61.31
Average Fx After Medication (Hz)	171.76	66.28
Fx Standard Deviation Before Medication (Hz)	2.68	2.18
Fx Standard Deviation After Medication (Hz)	3.16	2.78
Average Qx Before Medication (%)	39.75	3.73
Average Qx After Medication (%)	39.77	3.38
Qx Standard Deviation Before Medication (%)	0.06	0.06
Qx Standard Deviation After Medication (%)	0.06	0.04
Qx Range Before Medication (%)	4.84	2.55
Qx Range After Medication (%)	4.54	1.09
Jitter Before Medication (%)	1.29	1.39
Jitter After Medication (%)	1.34	1.27
Shimmer Before Medication (%)	4.39	1.93
Shimmer After Medication (%)	5.96	2.84

An examination of the individual scores of the Parkinsonian mildly dysarthic group before and after medication showed no specific patterns. Gender differences exist before and after medication. The scores of the female subjects (TZS2, GEI13, PX9) were higher than male scores. Figure 39 shows the individual scores before and after medication in average Fx variable.

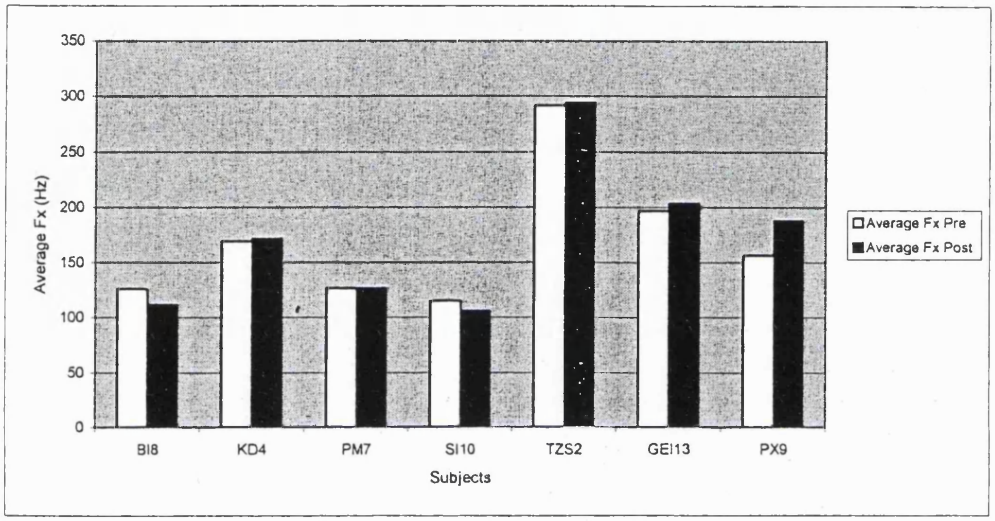

Figure 39. Individual scores before and after medication in average Fx variable.

Only in variable average Qx a small increase in individual scores after medication was observed which is in accordance with a small increase (0.02%) in the mean of the group. Since these increases are very small, their interpretation must be made with caution. It is safer to assume that medication seems to stabilise voice quality (at least in this task) rather than improve it.

5.3.2 Results of the main effect of medication and speaking task (reading and conversation) on voice

In review, the following variables were used in the current study in reading and conversation:

- Mean fundamental frequency,standard deviation and range (DFx1, DFx2)
- Mean relative intensity, standard deviation and range (DAx1, DAx2)
- Mean closed vocal fold contact, standard deviation and range (DQx1).

Table 18 presents a summary of the electrolaryngographic results in reading and conversation including variables, descriptive data, and analysis of variance (ANOVA). The Parkinsonian mildly dysarthric group was compared to itself in the same number of voice variables as in the between group results. The resulting data were analysed using a two-way analysis of variance in which one variable of interest was medication and the other variable of interest was speaking task to observe their effects on aspects of voice. Repeated measures analysis of variance did not show any statistically significant differences in the Parkinsonian mildly dysarthric group before and after medication (N = 7) in any logarithmically transformed variable.

Table 18. Summary results including variables, descriptive data and anovas in the Parkinsonian group before and after medication (N = 7)

VARIABLE – SPEAKING TASK	DESCRIPTIVE DATA Comparison of the scores in the Parkinsonian group before and after medication	ANOVA
DFx1 Mean in Conv. (Hz)	- No pattern - Increased pre and post medication scores in PD females compared to males - A large decrease in the score of the older male subject BI8 (74.2) after medication compared to other males scores	$p > 0.05$
DFx1 Mean in Read. (Hz)	- No pattern - Increased pre and post medication scores in PD females compared to males - A large decrease in the score of the older male subject BI8 (74.2) after medication compared to other males scores	$p > 0.05$
DFx1 SD in Conv. (Hz)	- No pattern - Decreased pre and post medication scores in PD females compared to males	$p > 0.05$
DFx1 SD in Read. (Hz)	- No pattern	$p > 0.05$
DFx1 90% Range in Conv. (Octave)	- Decreased scores in 5 of 7 PD subjects after medication - Increased pre and post medication scores in	$p > 0.05$

	PD males compared to females	
DFx1 90% Range – Read. (Octave)	- Increased scores in 5 of 7 PD subjects after medication - A large increase in the score of the older male subject BI8 (74.2) after medication compared to other males scores	p > 0.05
DFx2 Mean – Conv. (Hz)	- No pattern - Increased pre and post medication scores in PD females compared to males - A large decrease in the score of the older male subject BI8 (74.2) after medication compared to other males scores	p > 0.05
DFx2 Mean – Read. (Hz)	- No pattern - Increased pre and post medication scores in PD females compared to males - A large decrease in the score of the older male subject BI8 (74.2) after medication compared to other males scores	p > 0.05
DFx2 SD – Conv. (Hz)	- No pattern	p > 0.05
DFx2 SD – Read. (Hz)	- No pattern	p > 0.05
DFx2 90% Range – Conv. (Octave)	- No pattern - PX9 post medication score has a small increase (0.03)	p > 0.05
DFx2 90% Range – Read. (Octave)	- Increased scores in 6 of 7 PD subjects after medication (PX9 post medication score was decreased)	p > 0.05
DAx1 Mean – Conv. (dB)	- No pattern	p > 0.05
DAx1 Mean – Read. (dB)	- No pattern	p > 0.05
DAx1 SD – Conv. (dB)	- No pattern	p > 0.05
DAx1 SD – Read. (dB)	- No pattern	p > 0.05
DAx1 90% Range – Conv. (dB)	- No pattern	p > 0.05
DAx1 90% Range – Read. (dB)	- No pattern	p > 0.05
DAx2 Mean – Conv. (dB)	- No pattern	p > 0.05
DAx2 Mean – Read. (dB)	- No pattern	p > 0.05
DAx2 SD – Conv. (dB)	- No pattern	p > 0.05
DAx2 SD – Read. (dB)	- No pattern	p > 0.05
DAx2 90% Range – Conv. (dB)	- No pattern	p > 0.05
DAx2 90% Range – Read. (dB)	- No pattern	p > 0.05
DQx1 Mean – Conv. (%)	- Decreased scores in 5 of 7 PD subjects after medication - PX9 post medication score has a large decrease (10%)	p > 0.05
DQx1 Mean – Read. (%)	- Decreased scores in 5 of 7 PD subjects after medication - PX9 post medication score has a large decrease (11%)	p > 0.05
DQx1 SD – Conv. (%)	- No pattern	p > 0.05
DQx1 SD – Read. (%)	- No pattern	p > 0.05
DQx1 90% Range – Conv .(%)	- No pattern	p > 0.05
DQx1 90% Range – Read. (%)	- No pattern	p > 0.05

Table 19 below shows summary statistics of the variables measured during reading and conversation (means and standard deviations) before and after medication.

Table 19. Summary statistics of the variables measured during reading and conversation in the Parkinsonian group before and after medication (N = 7).

VARIABLE – CONDITION	READING		CONVERSATION	
	Mean	SD	Mean	SD
DFx1 Mean Before Medication (Hz)	165.99	63.91	171.47	55.60
DFx1 Mean After Medication (Hz)	163.76	65.09	167.50	64.12
Standard Deviation DFx1 Before Medication (Hz)	0.08	0.01	0.11	0.03
Standard Deviation DFx1 After Medication (Hz)	0.08	0.02	0.11	0.03
DFx1 90% Range Before Medication (Octave)	0.62	0.13	1.09	0.39
DFx1 90% Range After Medication (Octave)	0.67	0.16	1.06	0.33
DFx2 Mean Before Medication (Hz)	170.87	65.80	179.21	53.48
DFx2 Mean After Medication (Hz)	165.57	63.29	172.77	65.34
Standard Deviation DFx2 Before Medication (Hz)	0.03	0.01	0.05	0.01
Standard Deviation DFx2 After Medication (Hz)	0.03	0.01	0.05	0.00
DFx2 90% Range Before Medication (Octave)	0.50	0.11	0.89	0.27
DFx2 90% Range After Medication (Octave)	0.58	0.12	0.89	0.22
DAx1 Mean Before Medication (dB)	59.64	7.02	57.66	6.92
DAx1 Mean After Medication (dB)	58.37	7.62	57.58	6.92
Standard Deviation DAx1 Before Medication (dB)	8.66	1.20	8.66	1.20
Standard Deviation DAx1 After Medication (dB)	8.57	0.97	8.57	0.97
DAx1 90% Range Before Medication (dB)	13.94	2.54	15.94	2.45
DAx1 90% Range After Medication (dB)	13.76	2.17	15.90	1.79
DAx2 Mean Before Medication (dB)	60.67	7.11	58.77	7.03
DAx2 Mean After Medication (dB)	59.40	7.74	58.69	6.95
Standard Deviation DAx2 Before Medication (dB)	3.70	0.31	3.70	0.31
Standard Deviation DAx2 After Medication (dB)	3.50	0.44	3.50	0.44
DAx2 90% Range Before Medication (dB)	11.99	2.12	14.99	2.59
DAx2 90% Range After Medication (dB)	12.01	2.09	14.51	1.95
DQx1 Mean Before Medication (%)	38.93	5.56	38.79	5.35
DQx1 Mean After Medication (%)	36.07	4.04	36.79	5.50
Standard Deviation DQx1 Before Medication (%)	4.99	0.98	6.11	1.59
Standard Deviation DQx1 After Medication (%)	4.95	1.33	6.37	1.87
DQx1 90% Range Before Medication (%)	14.49	3.37	18.31	5.31
DQx1 90% Range After Medication (%)	14.53	4.09	18.80	5.44

Careful observation of the individual scores revealed a pattern only in DQx1 mean variables in both reading and conversation. This pattern is in accordance with

the group means of these variables. The Parkinsonian mildly dysarthric group exhibited lower means after medication than before in reading and conversation (DQx1 mean). In sustained phonation this tendency was reversed but involved an increase of only 0.02% (Average Qx). The assumption that medication may affect negatively voice quality (lower DQx1 mean) as the individual scores seem to point out, needs further research. The sample size as well as the large decrease in PX9's post medication score in DQx1 mean variable in conversation may account for the aforementioned pattern. Figures 40 and 41 show the individual scores before and after medication in DQx1 mean variable in both conversation and reading.

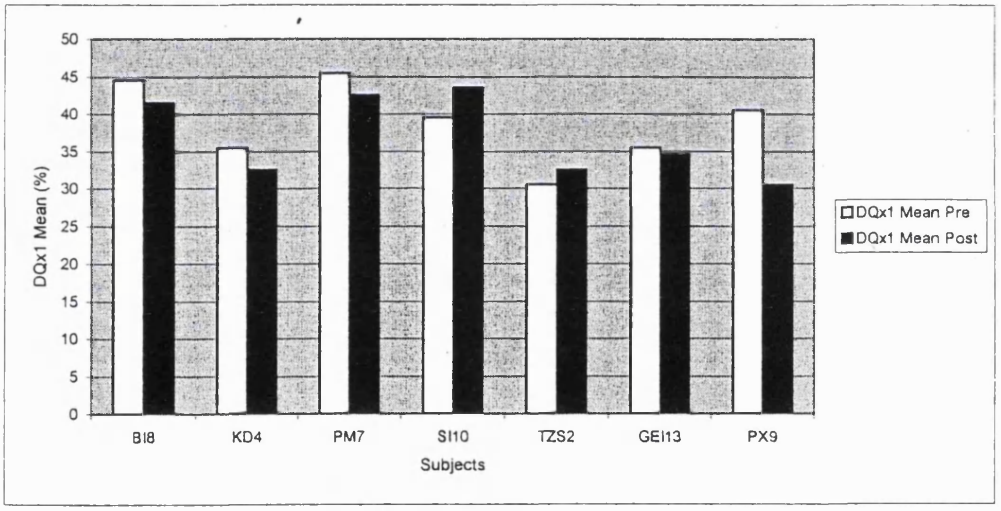

Figure 40. Individual scores before and after medication in DQx1 mean variable in conversation.

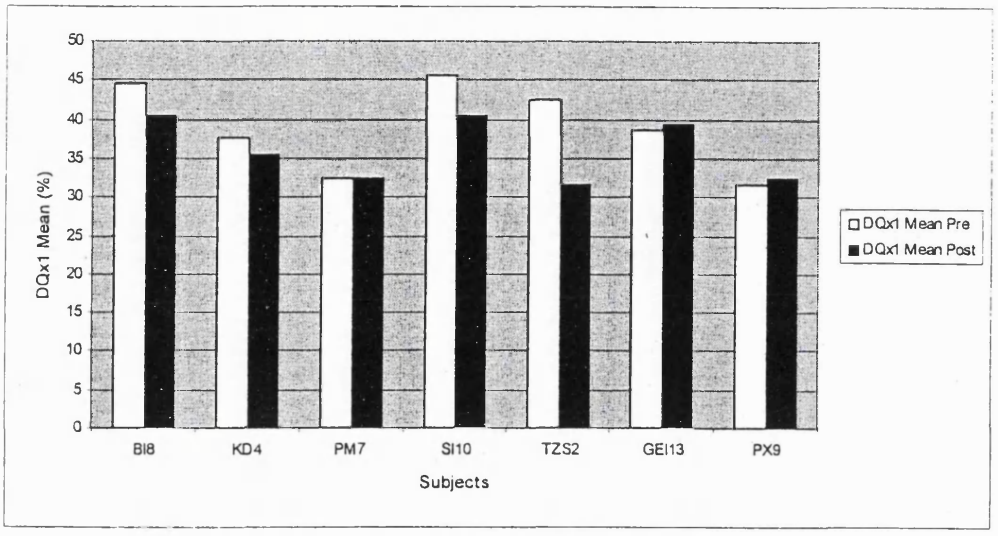

Figure 41. Individual scores before and after medication in DQx1 mean variable in reading.

As in chapter 4, gender differences were observed in the fundamental frequency variables in both conversation and reading (DFx1 and DFx2 mean variables). Female subjects' (TZS2, GEI13, PX9) scores were increased compared to male scores in both pre medication and post medication conditions. Figures 42 - 45 show the aforementioned gender differences in these speaking tasks.

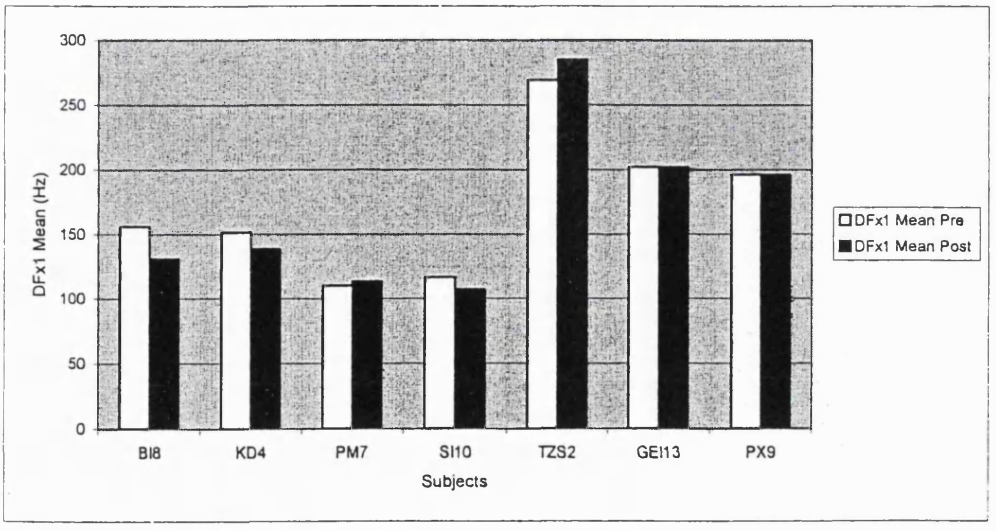

Figure 42. Individual scores before and after medication in DFx1 mean variable in conversation.

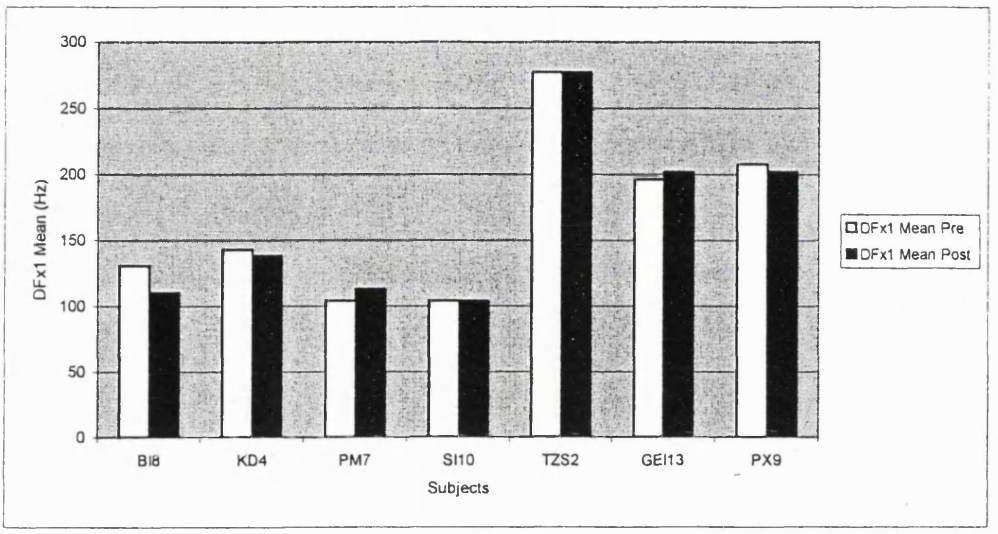

Figure 43. Individual scores before and after medication in DFx1 mean variable in reading.

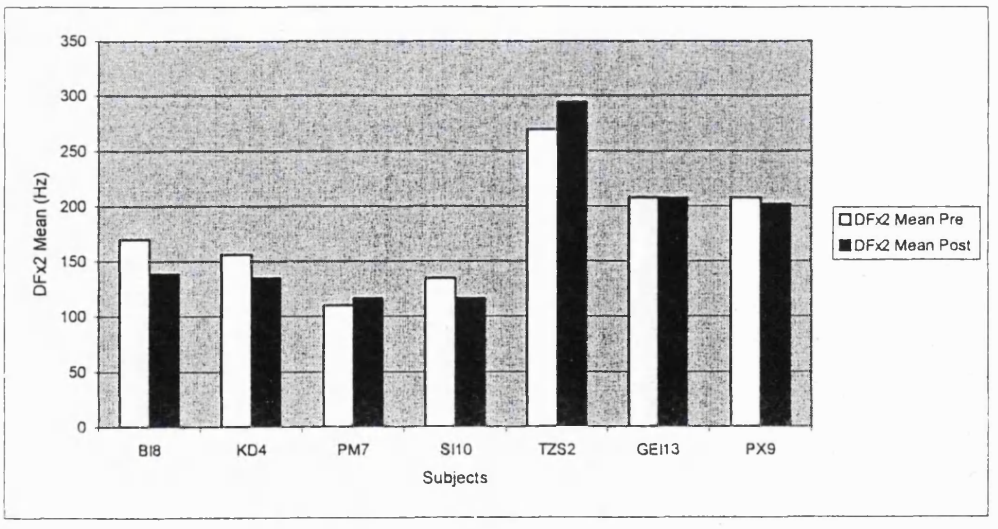

Figure 44. Individual scores before and after medication in DFx2 mean variable in conversation.

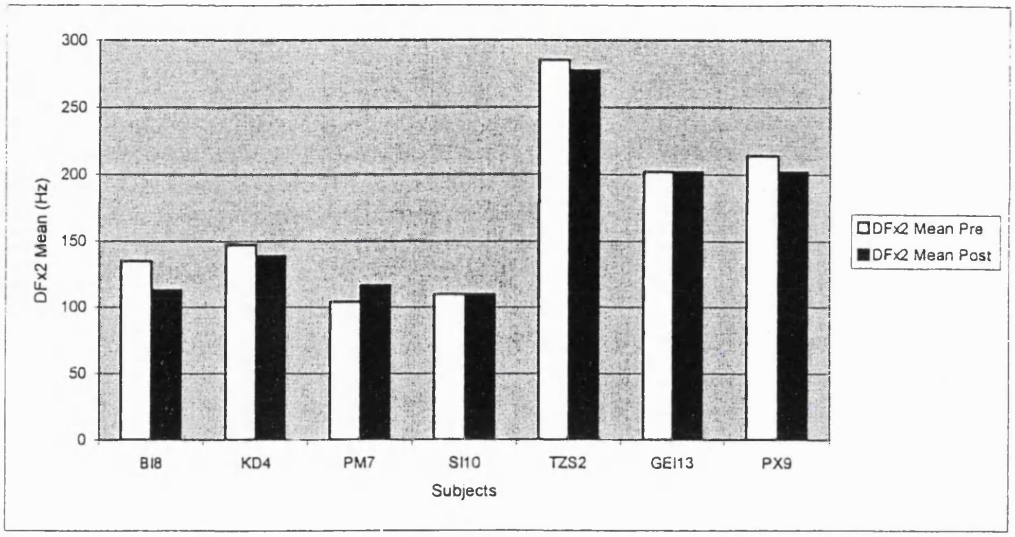

Figure 45. Individual scores before and after medication in DFx2 mean variable in reading.

Observation of the individual raw data shows that the after medication scores of the older male subject (BI8, 74.2 years old) had a large decrease in fundamental frequency in both reading and conversation (DFx1 and DFx2 mean variables) compared to other male scores (see the above figures). This decrease was 10 Hz in reading and 20 Hz in conversation. Further research is needed to clarify if medication affects fundamental frequency in older subjects.

Special attention must be given to subject PX9 who, apart from Parkinson's disease, exhibited dysphonia due to another cause. This subject exhibited large decreases after medication in the DQx1 scores (10%) in reading and conversation. Also, PX9 was the only subject whose after medication score was decreased in fundamental frequency range in reading (DFx2 90% range). The combination of her special pathology with the effect of medication may have caused these decreases.

In summary, from the observation of the individual scores it seems that medication has a differential effect in the Parkinsonian mildly dysarthric group. Factors such as age, gender, and dosage may interfere and cause these variations.

More research is needed to explore this issue. The present study shows that Parkinsonian subjects who are at the beginning of Parkinson's disease and who have received medication for the first time, are idiosyncratically affected by medication in the variables measured.

Along the same lines, the observation of the individual scores in reading compared to conversation before and after medication showed discrepancies between these tasks. Only, in DQx1 mean variable there was the same tendency of decreased scores with the group mean in Parkinsonian subjects after medication across speaking tasks. In all other variables, observation of raw data showed differences across speaking tasks. Repeated measures analysis of variance (ANOVA) was applied to identify if there was a main effect of speaking task (reading as compared to conversation) on voice[8]. In the Parkinsonian mildly dysarthric group (N = 7) before and after medication statistically significant differences were found. Table 20 below shows summary statistics of the Parkinsonian mildly dysarthric group before and after medication in the variables that were found to be significant in the main effect of speaking task on voice. Appendix R shows the results in reading as compared to conversation in all variables before and after medication.

[8] In two variables (DFx1 90% range and standard deviation DFx2) no normality of the distributions occurred. So, Wilcoxon paired sample tests were used.

Table 20. Summary statistics of the variables that were found to be significant in the main effect of speaking task on voice before and after medication (N = 7).

Variable	Task	Parkinson's Group Before Medication				Parkinson's Group After Medication			
		Mean	SD	Median	Range	Mean	SD	Median	Range
Standard Deviation DFx1 (Hz)	Conversation	0.11	0.03	0.10	0.08	0.11	0.03	0.11	0.06
	Reading	0.08	0.01	0.07	0.04	0.08	0.02	0.07	0.06
DFx1 90% Range (Hz)	Conversation	1.09	0.39	0.90	0.88	1.06	0.33	1.10	0.86
	Reading	0.62	0.13	0.59	0.40	0.67	0.16	0.72	0.45
Standard Deviation DFx2 (Hz)	Conversation	0.05	0.01	0.05	0.02	0.05	0.00	0.05	0.01
	Reading	0.03	0.01	0.03	0.02	0.03	0.01	0.03	0.02
DFx2 90% Range (Hz)	Conversation	0.89	0.27	0.79	0.72	0.89	0.22	0.89	0.53
	Reading	0.50	0.11	0.51	0.35	0.58	0.12	0.65	0.27
DAx1 Mean (dB)	Conversation	57.66	6.92	55.83	18.33	57.58	6.92	57.50	18.34
	Reading	59.64	7.02	58.61	17.78	58.37	7.62	58.06	21.67
DAx1 90% Range (dB)	Conversation	15.94	2.45	15.60	6.10	15.90	1.79	16.00	4.90
	Reading	13.94	2.54	13.40	7.40	13.76	2.17	13.00	6.30
DAx2 Mean (dB)	Conversation	58.77	7.03	57.50	18.89	58.69	6.95	58.61	18.89
	Reading	60.67	7.11	60.28	18.34	59.40	7.74	59.17	22.22
DAx2 90% Range (dB)	Conversation	14.99	2.59	15.30	6.80	14.51	1.95	14.70	5.90
	Reading	11.99	2.12	12.10	6.80	12.01	2.09	12.10	6.30
Standard Deviation DQx1 (%)	Conversation	6.11	1.59	6.99	4.08	6.37	1.87	6.74	5.17
	Reading	4.99	0.98	4.91	2.75	4.95	1.33	4.74	3.48
DQx1 90% range (%)	Conversation	18.31	5.31	21.00	12.10	18.80	5.44	19.30	14.40
	Reading	14.49	3.37	14.60	8.40	14.53	4.09	13.80	11.70

Ten variables were found that exhibited an overall effect of speaking task on voice. In eight out of these variables (SDDFx1, DFx1 90% range, SDDFx2, DFx2 90% range, DAx1 90% range, DAx2 90% range, SDDQx1 and DQx1 90% range) differentiation between reading and conversation was in both before and after medication conditions. Medication did not seem to differentiate the results across speaking tasks in the above variables. Also, comparisons between the mean group variables that were found to have a main effect of speaking task on voice showed

that DAx1 mean [$t(6) = -6.72$; $p < 0.01$] and DAx2 mean [$t(6) = -5.97$; $p < 0.01$] were significant only in the before medication condition. In general, since the individual scores do not show any pattern with the group means no conclusions can be derived from these findings and caution is needed in their interpretation.

In conclusion, the variables that were found to have an overall main effect of speaking task on voice were identical to both between group and within group analyses. The fact that there is a differentiation of speaking task on voice in both groups (Parkinsonian and control group) indicates that the voice of the Parkinsonian group is similar to that of people without the disease, as measured by the variables of the present study. In addition, this differentiation seems not to be affected by medication. However, this conclusion warrants further investigation due to the fact that no measurement of the speaking task on voice took place in the control group after 3-3.5 months, as it did in the Parkinsonian group due to variation of individual scores.

5.3.3 The main effect of medication on voice of the subject SB5 against the group of male subjects who took the same type of medication

Further analysis of individual data in the reading and conversation tasks for SB5 who stopped medication 1 month before the re-examination of his speech was made. It was considered important to see if the scores of SB5 differed from the scores of the group of the other 4 males who continued medication. For every experimental subject in the male Parkinsonian group who took levodopa as medication (SI10, PM7, KD4, BI8 and SB5), a z score difference was computed to determine the degree of the difference after medication as compared to the before medication condition. A z score denotes the number of standard deviations by

which a subject's score differs from the mean of the group and it is defined by the formula:

$$z = \frac{i - \mu}{\sigma}$$ [9]

In this study, the difference of performance in any variable after medication minus before medication in every subject, the mean of the differences of the group and the standard deviation of the differences of the group were employed. The z score difference was defined by the formula:

$$z\ difference^{10} = \frac{i\ after\text{-}before - \mu\ differences}{\sigma\ differences}$$

The relative intensity variables (DAx1 mean, DAx1 standard deviation, DAx1 range and DAx2 mean) showed a decrease of performance of the subject SB5 as compared to the 4 other male subjects of the group (BI8, KD4, PM7, SI10). In other words, the relative mean intensity difference (intensity after medication – intensity before medication) of the voice of subject SB5 is increased (large difference, the voice becomes quieter) and diverged as compared to the differences of the members of the group. This reduction ranged from 1.5 - 1.80 standard deviations in both reading and conversation. These differences however, did not seem to be perceptually apparent. Table 21 below, shows the z difference scores of the subject SB5 and their corresponding probability. Appendix Q shows the raw scores of the male subgroup who received levodopa in reading and conversation.

[9] i = individual score, μ = mean of the group, σ = standard deviation of the group
[10] in performance after medication minus before medication

Table 21. z differences in the logarithmically transformed scores of the subject SB5 as compared to the male experimental group that took the same type of medication (levodopa).

VARIABLE	READING		CONVERSATION	
	z Difference (sd)	P Value	z Difference (sd)	P Value
DAx1 Mean	-[11] 1.57	0.06	-1.63	0.05
DAx1 Standard Deviation	-1.74	0.04	-1.63	0.05
DAx1 Range	-1.78	0.04	-1.62	0.05
DAx2 Mean	-1.50	0.07	-1.67	0.05

[11] The negative sign denotes a worsening of performance in each score after levodopa medication. All p values are rounded to 2 decimal places.

Figures 46 and 47 below show the z difference in the scores of every subject of the male Parkinsonian group against the subject SB5 in DAx1 mean in reading and conversation, respectively.

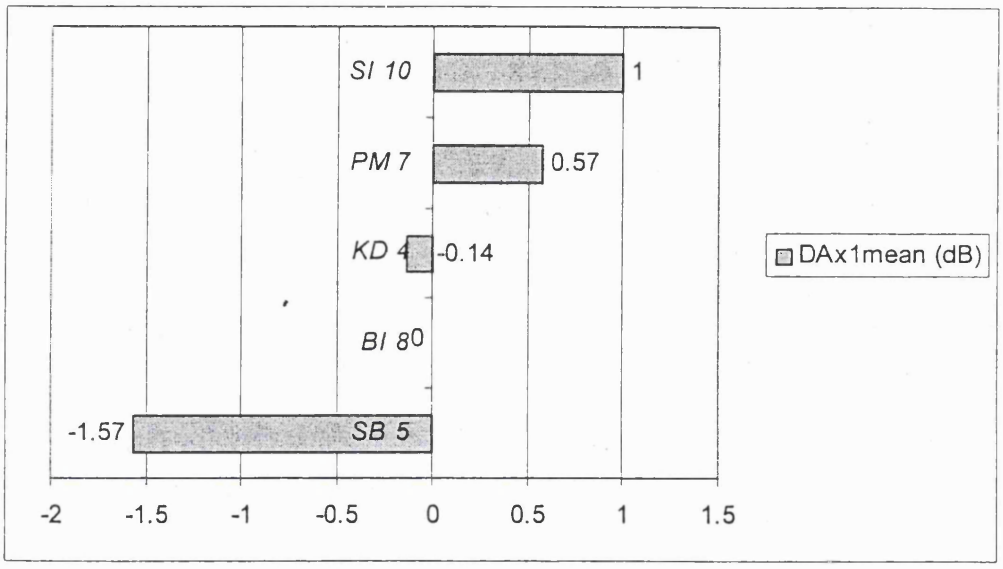

Figure 46. z difference expressed in standard deviations of DAx1 mean in reading.

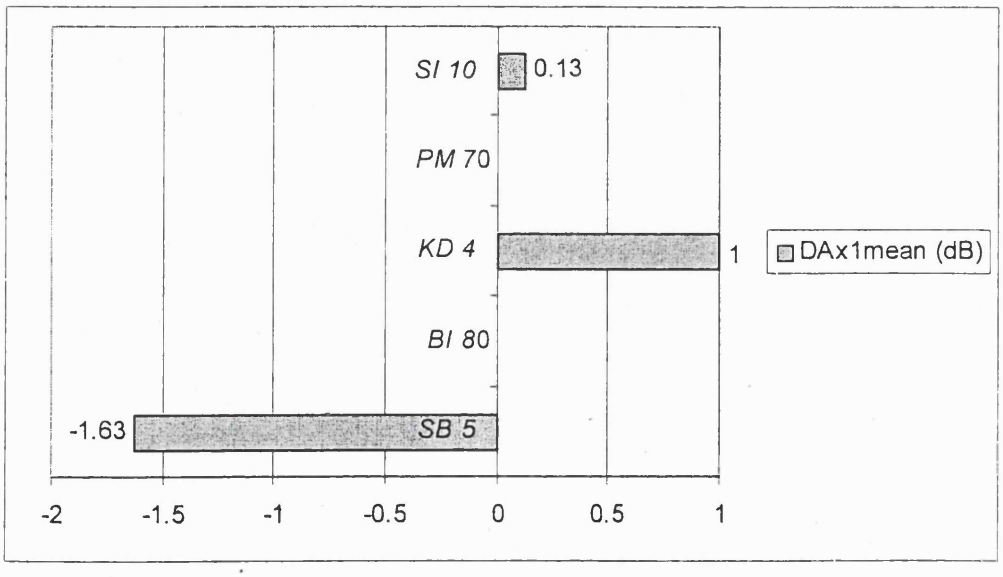

Figure 47. z difference expressed in standard deviations of DAx1 mean in conversation.

Figures 48 and 49 below show the z difference in the scores of every subject of the male Parkinsonian group against the subject SB5 in DAx1 standard deviation in reading and conversation, respectively.

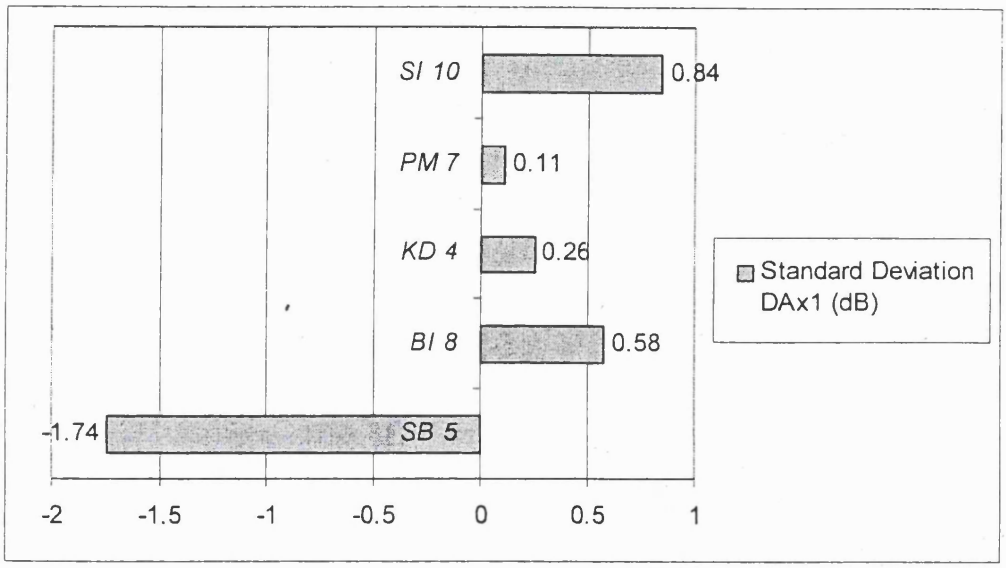

Figure 48. z difference expressed in standard deviations of standard deviation DAx1 in reading.

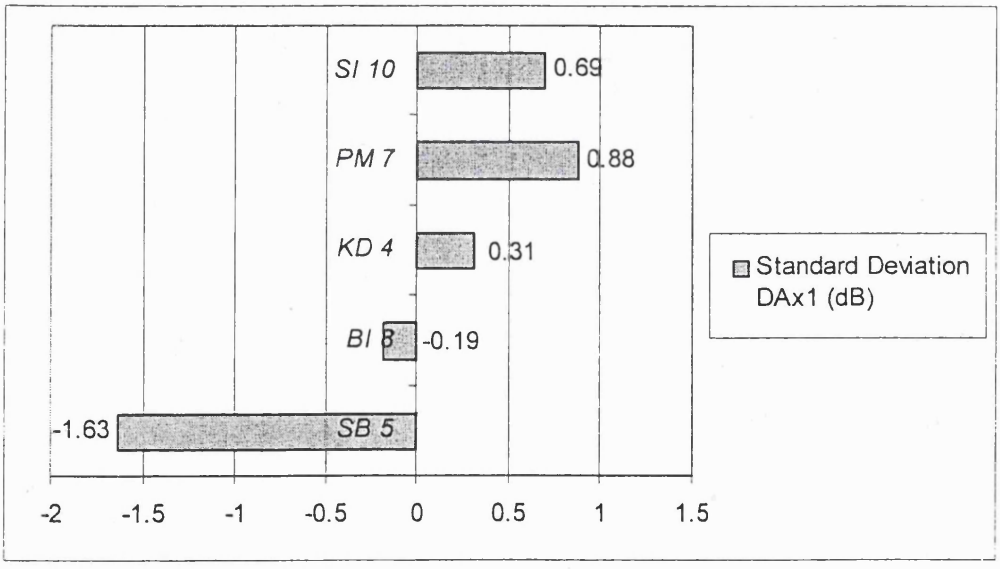

Figure 49. z difference expressed in standard deviations of standard deviation DAx1 in conversation.

Figures 50 and 51 below show the z difference in the scores of every subject of the male Parkinsonian group against the subject SB5 in DAx1 range in reading and conversation, respectively.

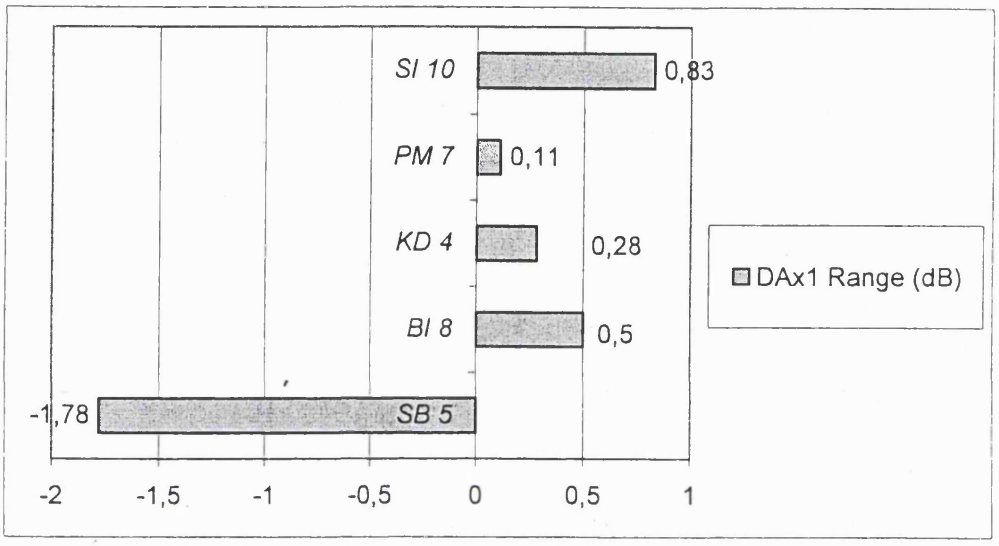

Figure 50. z difference expressed in standard deviations of DAx1 range in reading.

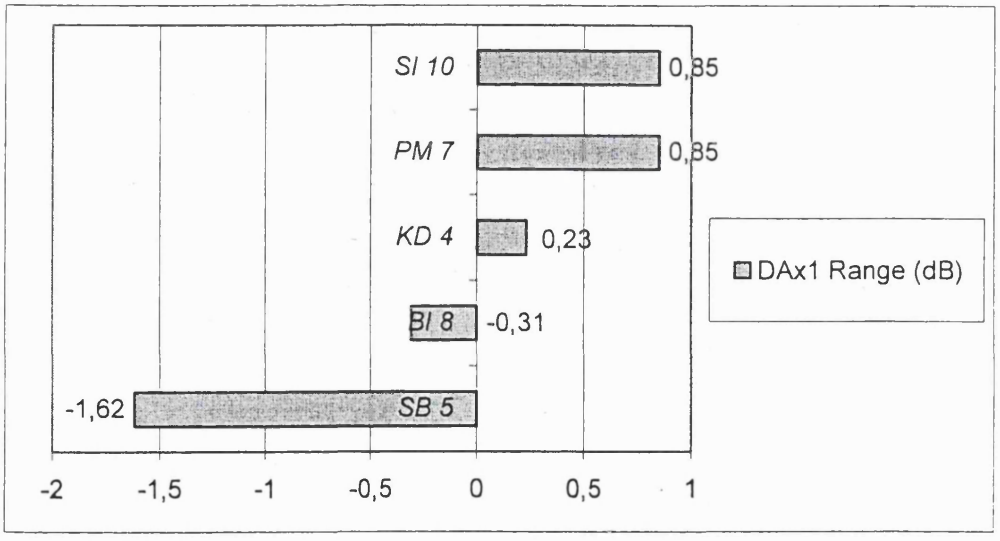

Figure 51. z difference expressed in standard deviations of DAx1 range in conversation.

Figures 52 and 53 below show the z difference in the scores of every subject of the male Parkinsonian group against the subject SB5 in DAx2 mean in reading and conversation, respectively.

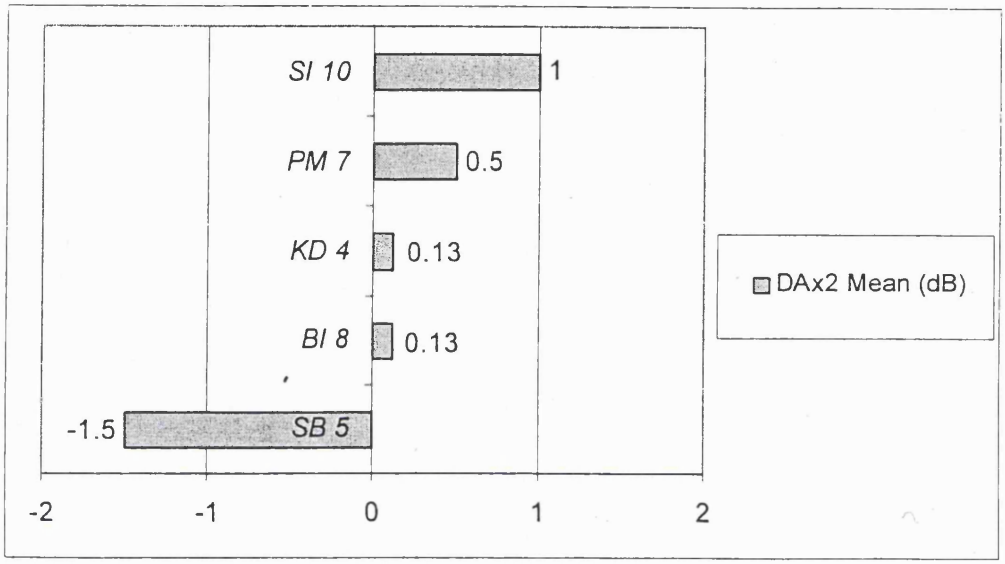

Figure 52. z difference expressed in standard deviations of DAx2 mean in reading.

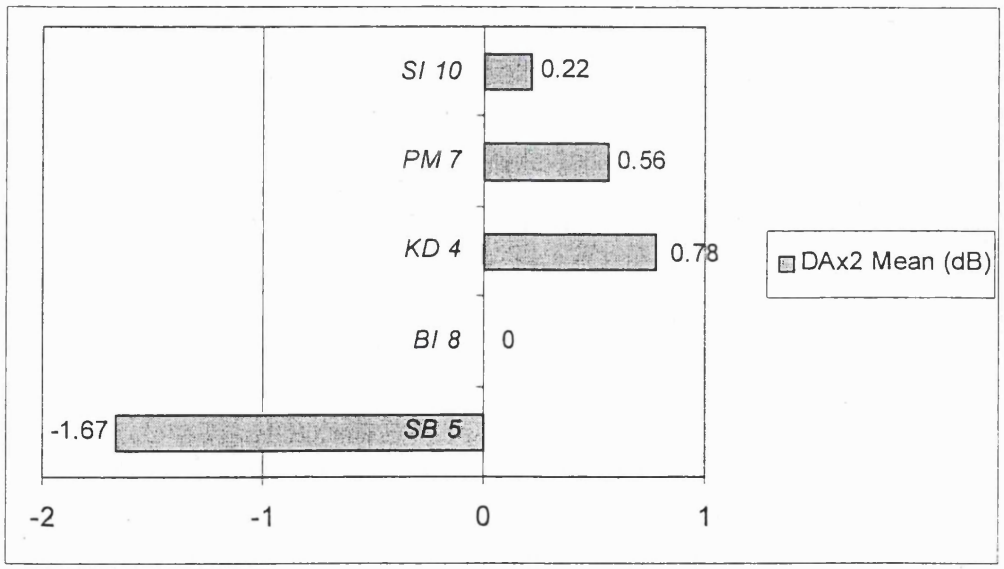

Figure 53. z difference expressed in standard deviations of DAx2 mean in conversation.

Examination of the individual data of subject SB5, who stopped medication one month before the re-examination of his voice against the group of male Parkinsonian subjects who continued levodopa medication, revealed decreases of performance (as expressed in z difference score) in relative intensity mean, standard deviation and range ($p = 0.04$-0.07). The other subjects of the group showed an increase of performance in the above variables but this was not statistically significant. This result points probably to a possible stabilisation effect of levodopa medication in relative intensity. Further research with more subjects is needed to prove this hypothesis.

5.4 Summary of the within group results

In summary, in the Frenchay Dysarthria Assessment (FDA) two mildly dysarthric Parkinsonian subjects showed no difference in their scores after medication (GEI13, SI10), four subjects showed an improvement (BI8, KD4, TZS2, PX9) and one subject showed a decline in performance (PM7). The improvement showed up primarily in the tongue area (increase in speed movement of the tongue) and the lips area (increased in speed movement of the lips). Medication seemed to have a variable effect in the laryngeal area during extended sustained phonation [(an improvement in two subjects in the laryngeal time and pitch subsections of the FDA (KD4, TZS2) and a decrease in two other subjects in the pitch, time and volume subsections of the FDA (PM7, PX9)]. The overall decline in one subject's performance (PM7) in the laryngeal and tongue sections of the FDA as compared to the other subjects, was probably due to his previous exposure to different drugs including levodopa and/or the progression of the disease itself.

No statistically significant differences in intelligibility were found when the group of the seven Parkinsonian mildly dysarthric subjects was compared to itself before and after medication. However, statistically significant intelligibility differences were found when the analyses were made without the subject PX9 who apart from Parkinson's disease exhibited dysphonia due to another pathology.

Electrolaryngographic measures were used to quantify aspects of voice in this group of seven subjects. Both the effect of medication and the effect of speaking task on voice were measured before and after medication. Observation of the individual scores showed individual variations in all variables except DQx1 mean variable. A decrease in the after medication scores in DQx1 mean variable was found in both reading and conversation even though this decrease did not reach statistical significance. However, this tendency warrants further investigation due to the small size of the sample. No statistically significant differences were found in the effect of medication on voice aspects. These variations may be due to different responses to medication that may have been caused by factors such as age, gender and dosage of medication. The present study shows that Parkinsonian mildly dysarthric subjects who are at the beginning of Parkinson's disease and who have received medication for the first time, are idiosyncratically affected by medication in the variables measured.

In the effect of speaking task on voice, the same variables that were found to differentiate reading from conversation in the between groups results, were found also in the within group results. Only two variables (DAx1 mean and DAx2 mean) that differentiated reading from conversation in the before medication condition did not show the same pattern in the after medication condition. However, the individual scores did not show any pattern and the validity of this finding is questionable.

CHAPTER 6. DISCUSSION

6.1 Incidence of dysarthria in early Parkinson's disease based on the results of the Frenchay Dysarthria Assessment

Factors that may contribute to different results of the reported incidence of dysarthria are the severity of Parkinson's disease at the time of assessment (neurological stage of the disease), the type of study (survey or clinical), the skills of the evaluator, possibly the medication at the time of assessment and the methods used for assessment. The majority of studies and reports have differences in one or all of the afore-mentioned factors. For example, two studies and one report included subjects with different neurological staging and medication during the dysarthria assessment (Hartelius & Svensson, 1994; Logemann et al., 1978; Scott et al., 1985). Also, the studies that employed a large number of subjects (more than 200 subjects) were either surveys (Hartelius & Svensson, 1994) or a combination of a survey and physical examination (Mutch et al., 1986) and their dysarthria assessment was based on either subjects' reports or neurological scales. In addition, researchers in the medical field have selected speech as a symptom among many other symptoms to include in their studies (Hoehn & Yahr, 1967; Mutch et al., 1986). Consequently, their results may differ with the results of those examiners specialising in the field of speech pathology, who focus primarily on speech symptomatology and who use different methods to assess dysarthria.

The reported incidence may also involve differences in the definition of dysarthria. Some medical specialists seem to distinguish dysarthria from voicing problems or "hypophonia" in Parkinson's disease (Hoehn & Yahr, 1967). In the current study, the definition of dysarthria follows the suggestions of other

researchers in the field of speech pathology who see dysarthria as a term for a speech disorder which includes voicing problems (Darley et al., 1969a; Duffy, 1995).

With the exception of one medical study (Hoehn & Yahr, 1967) which found that only 3% of the patients reported speech problems as initial symptoms of the disease, all other studies show high occurrence of dysarthria ranging from 45%-90% (Coates & Bakheit, 1997; Hartelius & Svensson, 1994; Logemann et al., 1978; Mutch et al., 1986; Scott et al., 1985). The low incidence (3%) of speech symptomatology in the Hoehn and Yahr study may be due to factors such as the general speech assessment and the inclusion of all forms of Parkinsonism in their subjects. Even though no direct comparisons between the reported incidence of the majority of studies and the results of the present study can be made due to differences in the number of subjects, the results of the present study fall within the aforementioned range. Eight out of thirteen Parkinsonian subjects were found to score lower in the Frenchay Dysarthria Assessment (FDA) compared to a group of matched pair controls.

Special attention should be given to the results of one study (Coates & Bakheit, 1997) because it is one of the few studies that examines an incidence of dysarthria based on both clinical examination and patients' reports. Similar to the present study but using a different neurological scale and intelligibility assessment, Coates and Bakheit aimed to identify the incidence of verbal communication disability[12] in Parkinson's disease. One third of patients were found unaware of their communication disability while half of patients (24/48 patients) were mildly affected (mild speech disturbances) and had no significant verbal communication disability (reduced intelligibility).

[12] Verbal communication disability was defined as the inability of the speech and language therapists to fully understand the speech of subjects (Coates & Bakheit, 1997).

Three aspects of the study by Coates and Bakheit should be noted. First, the reported incidence does not involve testing of voicing aspects. The incidence of verbal communication disability probably would be different if voicing were measured. Second, the authors supported the notion that the subjective views of patients about their speech problems may be misleading because they tended to overestimate or underestimate their speech problems. This tendency of the patients may probably be caused by physiological decrements of function due to normal ageing and/or differences in personality. The normal ageing process may be a confounding factor and it may complicate and distort the results of the dysarthria assessment. Great variability between subjects in speech performance has been reported as a result of the ageing process (Liss et al., 1990; Weismer, 1984a). Moreover, slowness of speech is considered an actual finding of the ageing process (Liss et al., 1990). Liss and her colleagues stated that ageing might involve speech processes very similar to those seen in a neurological disease. In fact, the sensory-motor performance of elderly people may be concomitant with neurochemical changes in the basal ganglia. The use of a control group matched in as many aspects as possible with the experimental group may be a solution to the potentially confounding effect of the normal ageing process on speech. Coates and Bakheit (1997) did not use a control group in their study. In the present study, a control group that was matched to the Parkinsonian group in age, gender and education was used to provide a clearer picture of the results regarding normal ageing.

Finally, all subjects (except two) in the study by Coates and Bakheit (1997) were on medication at the time they were examined. Some authors emphasise that the administration of dopamine therapy in Parkinsonian patients may have changed the character of dysarthria typically seen in such patients

(Duffy, 1995; Ludlow & Bassich 1984). If medication act positively (the patients' speech becomes better) then a lower incidence of dysarthria would be found. If it act negatively (the patients' speech become worse) then a higher incidence of dysarthria would be found. The results of the current study suggest that in the Frenchay Dysarthria Assessment (FDA) the medication had a positive effect on the speech of the majority of subjects. If this is the case, then Coates and Bakheit possibly reported a lower incidence than the incidence without medication and so, the actual incidence of dysarthria in their subjects should be higher. The type/dosage of medication and the individual's response to medication may play a crucial role in the incidence of speech symptomatology and these aspects need to be investigated in future studies.

Together, medication and neurological staging are factors that can undermine the results on incidence rates. If speech follows the other motor complications (dyskinesias) that take place some time after levodopa medication, then it is reasonable to hypothesise that speech will worsen. In fact, Marsden and Parkes (1976) reported peak-dose orofacial dyskinesias (1-2 hours after medication) that were accompanied by oromandibular dystonia and created difficulties in speech and swallowing. The authors emphasised that levodopa does not halt the progression of the underlying pathology in Parkinson's disease (neurological stage) and that after 3-5 years of medication the major manifestations of the disease reappear in 60% of their patients. The evidence by Marsden and Parkes shows the direct or indirect relationship of levodopa and neurological staging with speech and as a consequence with the reported incidence.

In conclusion, the incidence of mild dysarthria in the present study is within the range of the majority of studies that focused on speech

symptomatology. The role of medication on the incidence of dysarthria is not clear because all of the aforementioned studies reported incidence rates in medicated patients and/or different neurological status. Moreover, the incidence based on patients' reports is subjective in nature especially in studies where no control group is investigated. The clinical nature of the present study, the homogeneity of the sample on neurological staging, the unmedicated status of the subjects and the use of a matched control group, avoids the effect of the confounding factors on incidence rate. The results of the present study are considered a modest contribution to the understanding of the incidence of dysarthria when all of the aforementioned factors are excluded.

6.2 Profile of the mildly dysarthric subjects based on the results of the Frenchay Dysarthria Assessment

This section aims to discuss in depth those aspects of speech in which incidence of mild dysarthria was found. The Frenchay Dysarthria Assessment (FDA) was used to measure dysarthria in the present study (Enderby, 1983). The FDA measures dysarthria during maximum performance subtests of non speech activities (movement of lips and tongue without voicing), speech activities (repeated non sense /iu/ and CVCV /kala/), voicing (extended sustained phonation in the production of [a], intonation in singing a scale and loudness in counting numbers with increased loudness) (Leuschel & Docherty, 2000), and conversation. This test is detailed and elaborate in measuring the timing of independent areas in speech production (lips, tongue, laryngeal, respiration and palate) during speech and non speech movement. Based on maximum performance subtests, the FDA alone does not describe the nature of

speech errors in dysarthria but it could point to areas where the problem might appear.

In the present study, the general results of the FDA showed a longer timing of the articulators (lips and tongue) in most of the subjects and a shorter in duration sustained phonation in few subjects during individual tasks, but this did not occur during conversation tasks compared to their controls. The nature of the present study that included subjects in the beginning of Parkinson's disease (immediately after the neurological diagnosis) is probably responsible for this finding. These findings come in accordance with other reports (Darley et al., 1969b, 1975; Duffy, 1995; Parnell & Amerman, 1996), in which the individual movements of tongue and lips in hypokinetic dysarthria are accomplished slowly. One study (Chenery et al., 1988) reported mild disturbances in the movement of lips and jaw and moderate disturbances in the elevation of tongue together with the prominence of laryngeal problems. However, abnormalities in all areas of the FDA in conversation (lips, jaw, palate, laryngeal and tongue) have also been reported by Chenery et al. Because no report of disease duration and medication status has been given in this study, the inclusion of subjects with variable duration of disease might be responsible for these findings.

Only one study (Parnell & Amerman, 1996) examined Parkinson's disease during the preclinical period (5 years before the official neurological diagnosis). This study found slowness of volitional non speech movements and a restricted range of tongue movement during elevation in one Parkinsonian subject. The results of the present study support its findings and emphasise that dysarthria in the beginning of the disease will be manifested as a longer timing of the articulators during non speech and speech movements (fast alternate movement of [ui]) but not during running speech. So, slowness of movement

appears first as a distinguishing feature of hypokinetic dysarthria but it is not accompanied by a reduced range of movement in all subjects. Because other studies that reported subjects with a variable duration of disease and neurological status (Chenery et al., 1988; Darley et al., 1969a, 1969b) showed slow movements of lips, tongue and decreased extended sustained phonation (Canter, 1965a; Ludlow & Bassich, 1983), it seems that these are characteristics of dysarthria that appear in early disease state and continue to affect speech movement during the disease process. Later on, when the slowness of individual movements will be accompanied with a reduced range of movement in the articulators, the fast repetitive movements, a significant characteristic of hypokinetic dysarthria will appear (Duffy, 1995).

6.2.1 Between groups – Frenchay Dysarthria Assessment

This subsection aims to discuss the findings of the Frenchay Dysarthria Assessment (FDA) and the intelligibility testing between the Parkinsonian and the control groups. Out of the 8 subjects who exhibited a lower scoring in the FDA compared to their matched pair controls, in 6 the tongue movement was decreased in speed, in 5 there was a disturbed phonation and in 4 the lip movement was slow. In 4 subjects, problems in separate independent areas existed (lips, tongue or laryngeal) while in the remaining 4 subjects, 2-3 areas (lips, tongue and laryngeal) were affected simultaneously. As stated above, the lower scoring involved maximum performance speech and non speech tasks rather than the conversation task of the FDA. The Parkinsonian subjects completed the tongue and lips tasks in a longer time compared to their matched controls. In contrast, the laryngeal tasks (extended phonation of [a] sound) were completed in less time by 3 Parkinsonian subjects. The latter finding

supports the results of studies that have found a decrease in the duration of extended sustained phonation and so, less laryngeal stability (Canter, 1965a; Ludlow & Bassich, 1983).

Pathophysiologically, the slowness of movements of the articulators is probably related to the general neurological symptom of bradykinesia. Even though the terms akinesia, hypokinesia and bradykinesia have been used interchangeably to indicate loss of the ability to move (Marsden, 1989), bradykinesia and hypokinesia are employed in explaining the pathophysiological basis in the present study. The increased in time execution of tongue and lip movement of Parkinsonian subjects in the beginning of the disease may denote a slowness of movement (bradykinesia) while the decreased in time voicing during prolongation of the [a] sound may be explained by hypokinesia (reduced range of movement).

However, it is questionable whether this longer timing in all areas necessarily leads to articulatory problems at this stage of the disease. It might be more reasonable to say that slower movement as described by the Frenchay Dysarthria Assessment (FDA) could increase the possibility of speech errors when the rate of speech stays the same. Accordingly, Ackerman and Ziegler (1991) reported that the reduction of articulatory precision in consonant production in Parkinson's disease is accompanied by normal rate at the expense of movement excursion.

Disturbed tongue and lip movement increases the possibility of articulatory errors. Inadequate tongue elevation for complete closure in stop-plosives, fricatives and affricates was found in subjects with a variable course in Parkinson's disease (Canter, 1965b; Logemann & Fisher, 1981; Parnell & Amerman, 1996). Canter (1965b) reported that the incidence of tongue

involvement was higher than lip involvement in diadochokinetic rates. The prominence of slower tongue movement in the FDA of the present study does not support the findings by Logemann et al. (1978) or the report by Critchley (1981) that laryngeal and respiratory dysfunctions appear first in hypokinetic dysarthria. However, etiological, methodological differences and sample size variations might be responsible for these discrepancies.

Etiologically, these studies mentioned subjects with both idiopathic and postencephalitic Parkinsonism. Postencephalitic Parkinsonism was reported by Hoehn and Yahr (1967) to have a high occurrence of speech symptomatology (12/44 cases) as compared to idiopathic Parkinson's disease (7/183 cases). Methodologically, the lack of a normal control group in the study by Logemann et al. (1978) might account for the reported prominence of laryngeal involvement. Weismer and Martin (1992) supported the notion that if the study by Logemann et al. had investigated the speech of normal geriatric control subjects which could have been matched with the experimental group, similar voicing problems with the Parkinsonian group would have been found. In acoustic studies, ageing seems to be associated with a loss of voicing control (Liss et al., 1990; Weismer, 1984a) at least in the initial syllable position. Perceptually, normal ageing has been found to be associated with increased breathiness, vocal tremor, reduced loudness, slower speech rate and less precise articulation (Linville, 2000). These observations lead to the conclusion that the high incidence of laryngeal dysfunction that was found by Logemann et al. might indeed be lower at the beginning of the disease. Differences in the sample size between these studies and the present study may also be responsible for the reported discrepancies. The sample of the present study is too small (N = 13) to make generalisations about the prominence of tongue involvement in Parkinson's disease. In addition,

in the present study the Parkinsonian subjects were seen in the very beginning of the disease process compared to all the other studies that examined speech in Parkinson's disease.

Finally, in the study by Logemann et al. (1978) there was no information about the duration of the disease, the duration of medication intake before the speech assessment or the neurological stage of the disease. As has already been mentioned, these factors may be important because they may be related directly or indirectly to speech and voice. Almost all of the studies that focused on hypokinetic dysarthria, either included subjects with different duration of the disease, neurological status, or involved subjects under medication at the time of assessment (Ackerman & Ziegler, 1991; Baker et al., 1998; Canter, 1965a; Chenery et al., 1988; Fox & Ramig, 1997; Gamboa et al., 1997; Hammen & Yorkston, 1996; Holmes et al., 2000; Kent et al., 1994; Kent & Rosenbek, 1982; LeDorze et al., 1998; Ludlow & Bassich, 1983, 1984; Ludlow et al., 1987; Penner et al., 2001; Weismer, 1984a; Zwirner & Barnes, 1992).

In conclusion, it is uncertain that the problems that were identified by the Frenchay Dysarthria Assessment in the present study could lead to speech errors. In other words, a longer time to produce a movement of the tongue might not be enough to create an error. If however, a slower movement of the tongue is accompanied by a regular rate of speech, then the possibility for errors is increased. In hypokinetic dysarthria, variability of rate as a main speech symptom is well established (Adams, 1997; Chenery et al., 1988; Hammen & Yorkston, 1996; Ludlow & Bassich, 1983, 1984; Netsell, 1986; Scott et al., 1985; Zwirner & Barnes, 1992) and thus no generalisations could be made on the possibility of articulatory errors.

6.2.1.1 Intelligibility between groups

It is well known that imprecise articulation is highly correlated to speech intelligibility (Chenery et al., 1988; Darley et al., 1969a). A lower intelligibility score may be reflected in an increased number of speech errors. The perceptual impression of the researcher during the completion of the history form was that no major problems in intelligibility existed in either group of subjects. The results of the intelligibility testing were similar to the researcher's impressions. The Parkinsonian group and its matched pair control group had the same intelligibility mean (97.68% for the Parkinsonian group and 97.67% for the control group) and the individual scores were 90% and above.

The nature of errors in intelligibility assessment is susceptible to variability because of the lack of equivalence in each set of words given during testing. Every subject was exposed to the same number of words (70) but not to the same set of words. The finding that both groups had more or less the same number of intelligibility errors does not necessarily mean that the groups exhibited similar profiles. Even though the number of errors was similar, it is not clear if the nature of errors in both groups was the same. It is possible for a category of phonetic contrasts to be different between the two groups. Unfortunately, no such comparison can take place in the present Greek intelligibility list because of the heterogeneity of phonetic contrasts to which each subject was exposed (different sets of words).

In general, the findings of the present study for intelligibility focus on the number of errors and not the nature of errors. Another study that could use a list of intelligibility (preferably standardized) in which all subjects could be exposed to the same number of phonetic contrasts might reveal differences in the nature of errors between the two groups.

Intelligibility has no straightforward relationship with duration of disease. The smaller duration of disease in the present study might not be responsible for the high intelligibility percentages of the Parkinsonian subjects. In the hypokinetic dysarthria of Parkinson's disease, there are few studies that have measured intelligibility in Parkinson's disease. Coates and Bakheit (1997) used the Assessment for Intelligibility in the Dysarthric Speech (AIDS) (1981a) as an assessment tool and found an intelligibility mean percentage of 84.2% in 48 patients with Parkinson's disease (mean duration of disease 6.7 years). Kent et al. (1994) reported intelligibility scores in 31 males and females having hypokinetic dysarthria with different duration of disease. Only, five subjects out of the overall sample had a lower duration of disease (1-4 years). Their intelligibility scores from Kent et al. are presented in table 22.

Table 22. Intelligibility scores of Parkinsonian subjects with duration of disease of up to 4 years.

Subjects	Gender	Duration of the Disease (years)	Intelligibility
BPM	Male	1	96.7%
OPM	Male	1	89.0%
APM	Male	3	90.1%
PPM	Male	4	91.8%
GPM	Female	4	94.7%

A careful observation of the table shows that the short duration of disease does not necessarily lead to a higher intelligibility score. Persons with one year of duration of the disease exhibit almost 8% difference in their intelligibility scores. So, it is still not clear how duration of disease is related to intelligibility. Some studies have reported a negative correlation (Coates & Bakheit, 1997;

Metter & Hanson, 1986; Netsell, 1986) while one study reported the opposite (Hartelius & Svenson, 1994). The results of the present study show that in the beginning of the disease, the Parkinsonian subjects obtained high intelligibility scores.

6.2.2 Within group (medication) – Frenchay Dysarthria Assessment

The medication had an overall effect on the individual movements of the articulators as described by the Frenchay Dysarthria Assessment (FDA) in 5 out of the 7 Parkinsonian mildly dysarthric subjects. Four subjects exhibited a positive effect while one subject exhibited a negative effect. The same scoring was found in 2 subjects before and after medication, from which one subject (GEI13) used a different type of medication (dopamine agonist, "Mirapexin"). The Parkinsonian subjects who were affected by medication, exhibited the following differences in independent areas of the Frenchay Dysarthria Assessment:

- An improvement in the score of the tongue area in 4 out of 5 subjects. In the subject PM7, tongue movement before medication had been normal but worsened after medication.
- An improvement in the score of the lips area in 3 out of 5 subjects. The remaining two subjects scored equally after medication as compared to before medication.
- An improvement in the score of the laryngeal area in 2 out of 5 subjects. Two other subjects scored lower after medication as compared to before. Finally, one subject scored the same before and after medication.

Table 23 below shows the areas of the FDA that were improved after medication, the combination of medication and the age of the subjects.

Table 23. Age and combination of medication in the subjects who exhibited a positive effect of medication on the Frenchay Dysarthria Assessment.

Subjects	Age	Medication
Tongue Area		
KD4	66.1	Madopar 1 ×3 Akineton ½ ×2 Symmetrel 1×2
BI8	74.2	Madopar ¼ ×3 Akineton 1×1
SB5	79.9	Madopar 1 ×3
TZS2	64.8	Madopar 1 ×3 Akineton 1 ×3 Symmetrel 1×3
PX9	63.8	Madopar 1 ×3 Akineton ½ ×3
Lips Area		
BI8	74.2	Madopar ¼ ×3 Akineton 1×1
TZS2	64.8	Madopar 1 ×3 Akineton 1 ×3 Symmetrel 1×3
PX9	63.8	Madopar 1 ×3 Akineton ½ ×3
Laryngeal Area		
KD4	66.1	Madopar 1 ×3 Akineton ½ ×2 Symmetrel 1×2
TZS2	64.8	Madopar 1 ×3 Akineton 1 ×3 Symmetrel 1×3

In general, the combination of medication in the present study resulted in an improvement in the scores of the Frenchay Dysarthria Assessment (FDA) predominantly in the tongue area. Increases in the speed and rhythm were found in the lateral movement of tongue and the elevation of tongue. Also, the sealing of the lips and the appearance of the lips at rest were found to be positively affected by medication. In the laryngeal area however, the results were variable.

This might be due not only to specific indiosyncracies of the subjects (for example, PX9 had undergone parathyroid surgery), but also to the nature of specific voicing tasks of the FDA (extended phonation, singing a scale and counting with an increasing volume). The lack of studies that measure speech and voice before and after medication using the FDA prohibits any further conclusions.

The combination of levodopa-benserazide (Madopar) and anticholinergics (Akineton, Symmetrel) seems to be the most effective agent to describe the positive effect on the scores of the FDA. Anticholinergic drugs alone (Artane, Cogentin, Akineton and Symmetrel) do not seem to improve speech (Critchley, 1981). Their use is based on their ability to reduce tremor in the limbs, block the action of acetylcholine (Ach) and create a balance between dopamine and acetylcholine (Hermanowicz, 2001; Koller, 1992; Oertel & Quinn, 1997; Wills, 1998). Levodopa-benserazide (Madopar) seems to be the agent responsible for the favourable effect on tongue and lip movements.

As has been stated before, pathophysiologically, the results of the Frenchay Dysarthria Assessment (FDA) in the present study are probably related to the neurological symptom of bradykinesia (slowness of movement) that appeared as an early symptom of the disease together with tremor in all subjects. Slowness of movement is considered as the most important symptom in the diagnosis of Parkinson's disease (Hughes et al., 1992; Marsden, 1994; Tetrud, 1991). The general physical slowness of movement in the Parkinsonian subjects, might be responsible for the slowness in the individual movements of the articulators in the present findings of the FDA. This assumption confirms the clinical impression of the consultant neurologist that bradykinesia is responsible for dysarthria at this stage of Parkinson's disease. Madopar alleviated this

slowness of movement in both the limbs (as reported by the patients) and the isolated movements of the tongue and lips and less in the laryngeal area of the FDA. In the beginning of the disease, levodopa seems to have a beneficial effect on the individual movement of the articulators as measured in the FDA, but its role on the voicing tasks of the FDA is not so clear.

6.2.2.1 Intelligibility within group

As in between groups, medication showed a neutral effect on speech as measured by the "In Speech" subsections of the Frenchay Dysarthria Assessment (FDA) in the within group analysis. The scores of the FDA were normal before and after medication. Intelligibility as an index of communicative performance in the present study was improved. Only in one subject (PX9) with the laryngeal pathology, the intelligibility score after medication was lower than before medication. The mean intelligibility scores were statistically increased in the six remaining Parkinsonian mildly dysarthric subjects after medication. Even though no inferences about the nature of decreased intelligibility errors after medication can be made, some assumptions can be given about the number of intelligibility errors (after medication as compared to before medication condition). Before medication, the number of total errors that were found by both listeners was 21 compared to 12 (after medication).

These findings support the findings by Metter and Hanson (1986) that an improvement in intelligibility occurred when their patient was "on" levodopa medication. The current study supports the findings of studies that showed beneficial effects of levodopa on intelligibility as proven by an improvement in labial musculature (Leanderson et al., 1971; Nakano et al., 1973) and do not support the findings of other studies that did not show intelligibility improvement

(Gentil et al., 1998; Gentil et al., 1999). The intelligibility scale in the latter studies was general (not detailed examination) as a part of a neurological scale (UPDRS with scale of severity from 1 to 4). Also, the Parkinsonian subjects in these studies exhibited a moderate disease involvement and it is possible that after some years the response to medication was not consistent.

There are a number of reports in the literature that relate intelligibility with articulation or imprecise consonants (Canter, 1965b; Chenery et al., 1988; Darley et al., 1969a; Weismer & Martin, 1992). Canter (1965b) correlated the clarity of articulation with tongue movement (tip and back). If increased intelligibility after medication is due to more precise articulatory movements of the Parkinsonian dysarthric subjects, then it is possible that in the present study the higher scoring in the Frenchay Dysarthria Assessment subtests of the movements of the tongue followed by the lips and larynx, produced such increase. Further research is needed to correlate the isolated movements of the tongue area in the FDA with intelligibility scores in order to prove this hypothesis.

However, caution to the interpretation of the within group statistically significant results is needed due to a number of factors. First, all Parkinsonian subjects exhibited more than 90% intelligibility before medication. Second, the Greek word list (as all word identification tests) assesses intelligibility ignoring the context, and so it is not predictive of real-world communication situations (Hustad et al., 1998). Finally, no piloting of the Greek word list took place and weaknesses inherent to its development (differences in the frequency of contrasts between sets of words, differences in the overall number of each phonetic contrast category in each set) may limit the application of the statistically significant increase in intelligibility after medication.

In addition to the relationship of articulation to intelligibility there is one study (Ramig, 1992) that examined the relationship between vocal function and perceptual measures of speech including intelligibility in Parkinsonian patients who had received intensive voice therapy. Even though high correlations of monotone and shaky voice (0.88 and 0.74 respectively) with speech intelligibility were found, differences in the neurological stage of Parkinson's disease exist between the present study and Ramig's study. Ramig's study employed seven subjects with severe mobility problems implying an advanced neurological stage of Parkinson's disease. In the present study, it is possible that phonation (laryngeal section of the FDA) is not the area that improved intelligibility as shown by the small number of subjects who improved after medication in the laryngeal section of the FDA (n = 2). The areas that showed the most improvement in the FDA after medication were the tongue and lips. Consequently, errors that involved tongue and lips might respond better to medication and so, an increased intelligibility scoring would result. Further research is needed to prove this assumption.

6.3 Electrolaryngographic results in sustained phonation, reading and conversation between groups

6.3.1 Sustained phonation

Sustained phonation is a common task in the measurement of voice in neurological disorders. Kent et al. (1994) emphasised its frequent use in clinical voice evaluation. Despite its unnaturalness compared to conversation, simplicity and derivation of perceptual, acoustic or physiological measures make this task a preferential choice among researchers. Sustained phonation eliminates the effect of vocal tract configuration on the glottis and isolates the phonatory system

(Zwirner et al., 1991). Also, basic aspects of laryngeal function such as periodicity and amplitude can be derived from sustained phonation. In acoustics and electrolaryngography, the most common measures in sustained phonation involve the average fundamental frequency, standard deviation of fundamental frequency, jitter and shimmer. In this section, the results of the present study will be compared with the findings of similar acoustic and electrolaryngographic studies.

The between group results of the present study showed no significant differences in the group means of all variables. The average fundamental frequency was invariably found to be higher (although not always statistically significant) in many studies in the Parkinsonian group (Gamboa et al., 1997; Gerratt et al., 1987; Hanson et al., 1983; Hertrich & Ackerman, 1995; Kent et al., 1994; Ludlow & Bassich, 1983; Ludlow et al., 1983; Ramig et al., 1988). Statistical significance was reached in four studies (Gamboa et al., 1997; Hertrich & Ackerman, 1995; Ludlow & Bassich, 1983; Ludlow et al., 1983) while the non significant elevated fundamental frequency was found only in the male Parkinsonian group in another study (Kent et al., 1994). Two of the four studies reported that the statistically significant average fundamental frequency was found in their male subgroups (Gamboa et al., 1997; Hertrich & Ackerman, 1995). The other two studies (Ludlow & Bassich, 1983; Ludlow et al., 1983) despite their small number of Parkinsonian subjects (N = 7), they did not report any tendencies in the individual scores. In addition, bilaterality of symptoms and ambulatory problems indicate that the subjects in these studies were probably in an advanced stage of Parkinson's disease (in contrast to the present study that employed subjects in the early beginning of the disease). In the present study, observation of the individual data showed a tendency of decreased scores of

Parkinsonian subjects as compared to their controls in average Fx. The early stage of the disease may account for this discrepancy. Average Fx in sustained phonation may not be a sensitive measure in the beginning of Parkinson's disease.

Standard deviation of fundamental frequency (SDFo), as a measure of a long term phonatory instability has been found to increase even though no statistical significance was found (Zwirner et al., 1991). In the present study, no statistically significant differences between the Parkinsonian and control groups were found in SDFo. Individual scores of Parkinsonian subjects were higher compared to their controls which support the findings by Zwirner et al. (1991).

Jitter (cycle-to-cycle frequency perturbation) and shimmer (cycle-to-cycle amplitude perturbation) are also used very often to measure short-term variability in fundamental frequency and amplitude of the phonatory system (Baken, 1997). Many authors state that these measures are far from being standardised but because of their systematic examination in numerous studies they may be useful in clinical diagnosis (Heiberger & Horii, 1982; Horiguchi et al., 1987; Kent et al., 1994).

Increased jitter (either statistically significant or non statistically significant but elevated) was found by many acoustic and electrolaryngographic studies when Parkinsonian and control groups were compared (Gamboa et al., 1997; Gerratt et al., 1987; Hertrich & Ackerman, 1995; Holmes et al., 2000; Kent et al., 1994; Ramig et al., 1988; Zwirner et al., 1991). Statistically significant differences were found in three studies (Gamboa et al., 1997; Hertrich & Ackerman, 1995; Zwirner et al., 1991). In the Hertrich and Ackerman study, these differences were attributed only to the male subgroup while the remaining of the studies employed subjects with at least 6 years of disease duration who were at the stage II in

Hoehn and Yahr scale and were receiving levodopa. Holmes et al. found that statistically increased jitter is attributed to late Parkinson's disease rather than early Parkinson's disease. The present study found neither statistical significant differences between the two groups nor any individual tendencies, supporting Holmes et al. (2000) finding regarding the disease stage.

In many studies, shimmer was found to increase in the Parkinsonian group but did not reach statistical significance (Gamboa et al., 1997; Gerratt et al., 1987; Holmes et al., 2000; Kent et al., 1994; Ramig et al., 1988). One study found a statistically increased shimmer in the female Parkinsonian group (Kent et al., 1994). The results of the present study support the results of the aforementioned studies that found no difference in mean shimmer value among Parkinsonian and control groups. Comparison of the Parkinsonian subjects' scores with their matched pair controls revealed a tendency for decreased shimmer scores in 4 out of 6 Parkinsonian subjects. This tendency may reflect the different algorithm for the measurement of shimmer in the present study (Gold-Rabiner and cepstrum analysis from the corresponding to the Lx signal speech signal).

Qx was a variable used only in the present software for analysis (Speech Studio). Qx is described as a measure of voice quality (Abberton & Fourcin, 1997; Fourcin, 2000; Fourcin et al., 1995). Breathiness is reported to occur when the open phase of the vocal folds is extended in time as compared to the closed phase (Fourcin, 1981). In general, a lower Qx value reflects a voice with less stability in quality, as the mean percentage of time that the vocal folds are closed to the total period becomes smaller (a smaller time of closed vocal folds).

In the present study, the Qx mean variables that were found to be lower in the Parkinsonian group (although non statistically significant) compared to the

control group included the average Qx, the standard deviation Qx and the Qx range. The average Qx mean variable was the only variable to be consistently lower in the Parkinsonian group in both group means and individual scores. Although non statistically significant, the lower Qx value is probably a precursor of breathiness in the Parkinsonian group. A decreased average Qx variable confirms one study that reported similar tendencies and using similar methods. Hanson et al. (1983) reported a lower time of the vocal folds in the most closed period (15% of the entire cycle) in one Parkinsonian subject. This subject was in an advanced neurological stage of the disease and his voice appeared to be weak and breathy. Average Qx range was also found to decrease in the Parkinsonian group as compared to the control group but this involved only the mean values and not the individual scores.

Even though no statistical significance was found, the results of the present study in sustained phonation show that tendencies in Parkinsonian individual scores and groups means for elevated standard deviation of fundamental frequency, and decreased Qx were found in the beginning of Parkinson's disease. The results on the average Qx variable show that in the very beginning of the disease a lower closing time of the vocal folds during phonation may appear. More studies are needed especially on a longitudinal basis to observe if the average Qx variable is correlated to a perceived breathiness.

6.3.2 Reading and conversation

In the present study, fundamental frequency (DFx1 and DFx2), intensity (DAx1 and DAx2) and voice quality (DQx1) were measured during reading and conversation. The group results found non statistical significant differences in

these variables between the mildly dysarthric Parkinsonian group compared to the control group in both reading and conversation. The individual scores of 5 out of 6 Parkinsonian subjects in DFx1 and DFx2 in conversation were elevated compared to their controls, which is in accordance also with the group means of these variables. These tendencies did not appear in reading. In voice quality (DQx1), both in reading and conversation, comparison of individual scores and group means between Parkinsonian subjects and their matched pair controls, revealed lower scores and means for Parkinsonian subjects. These individual and group tendencies in the mean variables will be discussed against acoustic and electrolaryngographic studies that used similar measures.

Both DFx1 and DFx2 means (fundamental frequency of excitation) were found to be non statistically elevated in the Parkinsonian group compared to the control group in both speaking tasks. Statistically significant increased fundamental frequency during running speech was reported in three studies (Canter, 1963; Ludlow & Bassich, 1983, 1984). Although there was no information in these studies about the duration and neurological stage of the disease, inferences about bilateral symptoms probably imply that the Parkinsonian subjects were not at the beginning of the disease. Holmes et al. (2000) support the notion that statistically increased mean fundamental frequency is a characteristic of male subjects with late Parkinson's disease (13 years duration) rather than early Parkinson's disease. The findings in the present study of elevated fundamental frequency in conversation at the beginning of Parkinson's disease may show that this variable may be a precursor of the rigidity of the vocal folds. The same tendency was not found in reading (the individual scores did not present the same tendency with the mean) probably because this speaking task is more structured than conversation and the small

differences between the groups in DFx1 and DFx2 means may not be apparent in reading.

The group means of the standard deviations of the fundamental frequency of excitation and the individual scores did not reveal any pattern. Standard deviation of fundamental frequency and intensity during reading, especially the first, might reflect the voice disturbance (monopitch and monoloudness) in the Parkinsonian population (Gamboa et al., 1997). This measure has been used to show variability in the frequency of glottal opening and closing during phonation (Ludlow & Bassich, 1983; Ramig et al., 1988; Zwirner & Barnes, 1992). It is used as an index of phonatory stability and voluntary laryngeal movements (Zwirner & Barnes, 1992). Recent studies (Gamboa et al., 1997; Holmes et al., 2000) use it not only in sustained phonation but also in reading and monologue. Gamboa et al. found significant differences between a Parkinsonian group and a control group in the means of the standard deviation of fundamental frequency during reading. The Parkinsonian group in the study by Gamboa et al. exhibited a lower mean standard deviation of fundamental frequency compared to the control group. The present study does not support the findings of the study by Gamboa et al. Differences in duration of disease, neurological status and medication are probably responsible for the discrepancies between the two studies. Holmes et al. consider that statistically significant standard deviation of fundamental frequency (SDFo) is attributed in the female subgroup of late disease compared to early Parkinson's disease and controls.

Three studies (Canter, 1963; Gamboa et al., 1997; Ludlow & Bassich, 1984) reported significant differences in fundamental frequency range between Parkinsonian and control groups during reading of a sentence. Smaller mean fundamental frequency ranges were reported for the Parkinsonian groups as

compared to the control groups. The present study showed the same tendencies with the above studies, however, non statistically significant, in both the DFx1 90% range and the DFx2 90% range in reading. This trend did not appear in conversation.

The present study found no statistically significant differences in the variables DAx1 and DAx2 (intensity of excitation) between the two groups. Observation of individual scores revealed no pattern as well. These findings support the findings by Canter (1963) who reported that his groups overlapped in intensity. Similar results were found when Parkinsonian patients with early disease were compared to Parkinsonian patients with late disease (Holmes et al., 2000). However, one study (Fox & Ramig, 1997) showed different results. The means of the sound pressure level in reading and monologue of the Parkinsonian subjects were decreased statistically up to 4 dB compared to healthy controls.

Standard deviation of intensity was found not to be significant when the Parkinsonian group with early disease was compared to the Parkinsonian group with late disease (Holmes et al., 2000). It seems that the standard deviation of intensity is not a sensitive measure for variation during the disease process. The findings of the present study agree with the above observations. Intensity range in reading was found not to be statistically significant in the study by Canter (1963) which agrees with the present study. The present study however, showed a tendency for decreased scores of Parkinsonian subjects compared to their matched pair controls in DAx1 90% range and DAx2 90% range in conversation.

The means of the groups in DQx1 in both speaking tasks were non-statistically lower in the Parkinsonian group. The tendency of a lower DQx1 mean variable in all speaking tasks (sustained phonation, reading and

conversation) is accompanied by lower individual scores. As in sustained phonation, this finding (even though it did not reach statistical significance) might be a precursor of breathiness in the Parkinsonian group. Future studies may show if the DQx1 mean variable will reach statistical significance as the disease progresses. DQx1 90% range in conversation also was found to be lower in the Parkinsonian group compared to the control group, as both group means and individual scores have showed.

In conclusion, the between group results showed tendencies in the means of a number of variables. The tendencies that were confirmed by the individual scores were considered more valid to discuss the phonatory mechanism in the very beginning of Parkinsón's disease. The mean Qx variable in all speaking tasks was found consistently lower in the majority of Parkinsonian subjects. Pathophysiologically, these tendencies might be precursors of breathiness in the Parkinsonian group. Similarly, the elevated mean fundamental frequency in conversation that was found in the majority of Parkinsonian subjects might be a precursor of rigidity of the vocal folds. Also, smaller ranges in both fundamental frequency and intensity (as agreement of the individual scores with the group means have shown) were found and may relate to monopitch and monoloudness. However, these tendencies in ranges were dependent on the speaking task. From the above findings which did not reach statistical significance, the voice disturbance in the mildly dysarthric Parkinsonian group of the present study is not apparent, yet, but as the disease progresses it may become more evident.

6.4 Electrolaryngographic results in sustained phonation, reading and conversation within group (medication)

In sustained phonation, the within group results (before and after medication) showed no statistically significant differences between the group means in all variables. Observation of individual data in average Fx and standard deviation Fx variables showed no specific patterns. The results of the present study confirm the findings of other studies that reported no significant differences in mean fundamental frequency and standard deviation of fundamental frequency in sustained phonation (Daniels et al., 1996; Jiang et al., 1999). An interesting finding is the large increase (32Hz) in subject's PX9 score (subject with the laryngeal pathology) in average Fx variable after medication compared to all subjects and approaching other females' scores. Her increase in average Fx may be considered as a positive sign of improvement and not as a negative sign, given her low pre-medication score.

Jitter and shimmer showed no statistical significant differences in group means before and after medication, which is in accordance with the lack of any pattern after observation of individual data. The results on jitter support the findings by Jiang et al. (1999) where no significant differences after medication were found. In contrast, the results on shimmer do not support the findings by Jiang et al. who found a significant decrease in shimmer values in 11 out of 15 subjects in their study. Different algorithm of shimmer derivation might be possible reason for this discrepancy.

In variable average Qx a small increase in individual scores and group means after medication was observed. However, the increase is very small and probably indicates no alterations in the voice quality of the subjects after

medication compared to before medication. It is safer to assume that medication either plays no role or has a stabilizing role on this measure of voice quality.

There is only one study that has used electrolaryngographic measures in reading and conversation before and after levodopa medication (Daniels et al., 1996). The results of the present study support its findings that showed no statistically significant differences in all mean group variables before and after medication.

Observation of individual data showed some pattern in fundamental frequency ranges in both speaking tasks. These patterns were different across speaking tasks (in reading there was a tendency for increased scores after medication and in conversation the opposite), which may reflect the different demands of each speaking tasks on voice. Moreover, this differentiation across speaking tasks is a characteristic shown most notably in normal rather than dysarthric speakers (Leuschel & Docherty, 1996, 2001). Based on the above observation and the findings of the present study it is safer to say that in this early stage of the disease, voice is not yet affected.

A clear tendency of decreased individual scores in DQx1 mean in both speaking tasks after medication, in accordance also with the non-significant lowering of the within group means, may reveal a possible interesting area of research. The effect of medication (indifferent, stabilizing, or worsening) on voice quality (DQx1) needs further research.

Statistically significant differences in relative intensity variables were found when z differences expressed in standard deviations were compared between the subject SB5 (stopped medication after 2 months) and the group of levodopa medicated male subjects who continued medication. These findings may indicate that the combination of levodopa and anticholinergics (especially

levodopa) may act to stabilise the intensity of voice in the medicated male subgroup (all relative intensity variables were increased up to 1 standard deviation). These results support the findings by Metter and Hanson (1986) where the relative intensity was lower in one patient with Parkinson's disease in the "off" levodopa medication condition and was increased after levodopa administration. Further research is needed to examine the validity of this assumption and probably correlate the perceptual impressions of loudness decay in Parkinson's disease (Darley et al., 1969b) with intensity in subjects before and after medication. However, the results of subject SB5 compared to the male medicated subgroup should be taken with caution for two reasons. First, the small number of subjects does not allow for a generalisation of these findings. Second, the age of subject SB5 (the oldest experimental subject, 79.9 years) might be responsible for the z differences in intensity and not the type of medication, per se. The ageing effect in healthy individuals involves a decrease of function in speech (Liss et al., 1990) and voice (Linville, 2000). Linville (2000) states that increases in mean fundamental frequency and intensity variability with age have been found in males. Along the same lines, the individual scores of one male subject who was 74,2 years old (BI8), second older after subject SB5, showed a large decrease (20 Hz) after medication compared to the other male scores in mean fundamental frequency in both speaking tasks.

6.5 Gender differences

In the present study, gender differences were found in fundamental frequency in all speaking tasks. Both, Parkinsonian and control female subjects exhibited higher scores than males. These results are in accordance with studies

reporting higher fundamental frequency in females compared to males in normal voice production (Baken, 1997; Greene & Mathieson, 1997).

In voice production in Parkinson's disease, two studies (Hertrich & Ackermann, 1995; Gamboa et al., 1997) found increased mean fundamental frequency only in the male subgroup of their subjects compared to controls. In the present study, no such finding was observed. This may be due either to the early stage of the disease or to the small sample of the present study (4 male and 2 female Parkinsonian subjects completed the electrolaryngographic measures).

Observation of the individual data after medication did not reveal any gender specific patterns. Medication does not seem to differentiate between males and females regarding the variables examined in this study. Pre and post medication female scores in fundamental frequency remain increased compared to male scores.

6.6 Summary of the results and the discussion

According to the Frenchay Dysarthria Assessment (FDA), the Parkinsonian and the control group appeared to have differences in the individual movements of the articulators and not during the production of running speech. The number of errors in intelligibility testing was similar in both groups, with a slight increase of errors in the Parkinsonian group, while the nature of errors was not possible to be examined in the present study. Pathophysiologically, bradykinesia and tremor in the right limb were the predominant neurological symptoms for the group of Parkinsonian subjects. Also, the perceptual impression of the consultant neurologist was that bradykinesia (slowness of movement) is associated to dysarthria at this stage of

the disease. In the first impression, this assumption comes in contrast to reports that hypokinetic dysarthria is caused by rigidity of the laryngeal (Jiang et al., 1999; Kent, 1990), vocal tract (Kent, 1990) and respiratory musculature (Critchley, 1981). However, differences in neurological staging, duration of disease and medication status of the present study as compared to all other studies that investigated hypokinetic dysarthria could justify such an assumption. It would be reasonable to say that hypokinetic dysarthria in the very beginning of the disease appears as slowness of the articulators during volitional speech and non speech tasks (but not during running speech) and progresses to monopitch, monoloudness and reduced stress as the symptom of rigidity appears and affects the speech and voice musculature. Along the same lines, even though it was not statistically significant, an elevated fundamental frequency (in both group means and individual data) especially in the conversation task in the Parkinsonian group might be a precursor of rigidity in the laryngeal musculature. Similarly, a decreased Qx variable (in both group means and individual data) in all tasks in the Parkinsonian group might be a precursor of breathiness caused by rigidity.

In the after medication condition, important differences were found during the administration of the Frenchay Dysarthria Assessment (FDA), as compared to the before medication condition. These involved the individual movements of the tongue (lateral movement of tongue and the elevation of tongue) and lips (sealing of the lips and the appearance of the lips at rest) during non speech tasks that became more automatic and fast and less the individual movements of larynx. According to the FDA findings, levodopa seems to have a differential effect on the articulators rather than on voice.

Regarding phonatory instrumental measures (electrolaryngography) the effect of medication based on the findings of the present study is not clear. Statistically significant differences in relative intensity variables were found when one male subject who stopped medication was compared to the group of male subjects who continued medication. Even though the number of subjects prohibits generalisations of this finding, the combination of carbidopa/levodopa and anticholinergics probably acts to stabilise intensity of voice. This finding supports recent findings that levodopa improves vocal efficiency by increasing respiratory volume or amplitude of vocal fold vibration, or both (Jiang et al., 1999).

Observation of individual scores revealed tendencies in two mean variables in both speaking tasks: fundamental frequency ranges and DQx1. The way medication may affect these variables at this stage of Parkinson's disease needs further research, due to the small sample of the present study and in view of the lack of other electrolaryngographic studies examining the aforementioned area. In summary, from the observation of the individual scores, it seems that the effect of medication on electrolaryngographic measures is not clear at this stage of the disease. The findings of the present study seem to point out that Parkinsonian subjects who are at the beginning of Parkinson's disease and who have received medication for the first time, are idiosyncratically affected by medication.

6.7 Hypokinetic dysarthria in the progression of Parkinson's disease and explanation of the results in relation to medical, speech, and neuroscientific evidence

This section aims to discuss the findings of the current study in view of the progression of hypokinetic dysarthria in Parkinson's disease. A major assumption of this study and of most of the studies on hypokinetic dysarthria is that speech symptomatology is the effect of neurological symptomatology in Parkinson's disease (Darley et al., 1969a, 1969b; Duffy, 1995; Gentil & Pollak, 1995; Jiang et al., 1999; Kent, 1990; Ludlow & Bassich, 1984). However, the timing of the emergence of hypokinetic dysarthria is not yet known.

As has been stated in the literature review, in the general framework of neural speech production (Kent & Tjaden, 1997), the normal inputs from motor cortex, supplementary motor area, premotor cortex, somatosensory cortex and superior parietal lobule to the basal ganglia are processed to the thalamus. The excessive output from the basal ganglia due to the lack of dopamine results in an increased tonic inhibition of thalamic and cortical neurons (supplementary motor area, premotor cortex, Broadman area 44, cingulate cortex and motor cortex). The function of motor cortex as a working funnel for movement information from cortical and subcortical areas is disrupted due to insufficient information from the basal ganglia and thalamus. The result is a consequent insufficient output from motor cortex to the spinal cord, brainstem, α-motoneurons (i.e., a decrease in the frequency of their firing), premotor cortex, thalamic nuclei, striatum and red nucleus. Even though this explanation is theoretical in nature and the existence of simultaneous activity of different loops in the basal ganglia complicates the picture, there is no doubt that the neurological symptomatology (bradykinesia, rigidity and tremor) and speech symptomatology (rigidity) are the results of the

lack of dopamine. This fact is further enhanced by the existence of hypokinetic dysarthria and its differentiation when compared to the other types of dysarthria. The assignment of a sensory (impaired sensory trajectories) and motor role (Kent, 1990; Kent et al., 2000) or a sole motor role (Marsden, 1982, 1994) to the basal ganglia does not change this aforementioned fact. On a purely motor basis, for example, the muscle indices in the equations of constraint in the coordinative structures (action theory) may be set lower (without changing their fixed relationship) due to a reduced firing of α-motoneurons. Consequently, a reduced range of movement, as in hypokinetic dysarthria, can occur.

Bradykinesia and tremor were the predominant neurological symptoms in the sample of this study. In fact, bradykinesia (slow movement and difficulty initiating a movement) is reported to be the earliest symptom of Parkinson's disease (Scharre & Mahler, 1994). Its importance is also manifested by the fact that it is considered the most basic symptom to diagnose idiopathic Parkinson's disease (Hughes et al., 1992; Marsden, 1994; Tetrud, 1991). In addition, attempts of early diagnosis showed that the movement time for the upper limb (as a correlate of bradykinesia) was the most sensitive measure in detecting slight motor disturbances in early Parkinson's disease (Watts et al., 1991). Even though it is clear from many studies that hypokinetic dysarthria is the result of rigidity of movement in the speech musculature (Darley et al., 1969a, 1969b; Duffy, 1995; Gentil & Pollak, 1995; Jiang et al., 1999; Kent, 1990; Ludlow & Bassich, 1984), the effect of bradykinesia on speech has been overlooked.

In speech, the results of the present study showed that in the very beginning of Parkinson's disease bradykinesia may be the potential agent that affects the movement of the articulators. Slowness of limb movement as reported by the subjects of the present study was found to coincide with the

clinical findings of slowness in lips and tongue during non speech and speech movement. On a neuroanatomical basis, the structural proximity of oroface to leg and arm in the somatotopic organisation of putamen has been hypothesised in experiments on monkeys (Alexander & Crutcher, 1990). Marsden (1994) hypothesised that brainstem centers that control posture, locomotion and speech may be involved in the problems of posture, gait, and speech. Along the same lines, Kent et al. (2001) emphasised that overlapping neural systems control speech and non speech motor behaviours (especially in the limbs). On a neuropathological basis, similarities between limb and speech movement have been suggested (Cammicioli et al., 1998; Ho et al., 1999; Tetrud, 1991). However, limb movement and speech movement was considered not to have a direct relationship at least in advanced Parkinson's disease (Kent et al., 2001; Murdoch, 2001). Their functions were distinctive as proven by the fact that surgical and levodopa interventions have given a positive outcome on limb movement but not necessarily on speech (Kent et al., 2001). Moreover, some researchers suggest that there is a task dependency in speech movement of hypokinetic dysarthria (Connor & Abbs, 1991; Kent et al., 2000). They reported that in the proximity of 'leg', 'arm' and 'face' areas within the putamen and globus pallidus, there are cell groupings in each area that function in task-dependent movement effects. This was proven by the fact that differential effects were found in speech that were task dependent (visually guided jaw movements were found to increase in duration in jaw opening in non speech but not during a phrase production). These results were similar to visually guided arm movements.

In phonation, it has been stated in different studies that bradykinesia is responsible for the appearance of "hypophonic" speech (Baker et al., 1998;

Quinn, 1997). Marsden (1989) states that the clinical differences of bradykinesia and hypokinesia are not clearly manifested. In the present study, a decrease in extended sustained phonation in the laryngeal section of the Frenchay Dysarthria Assessment could be attributed either to bradykinesia or to hypokinesia. With the appearance of rigidity during the disease process, which may affect the laryngeal musculature, the phonatory impairment will become more evident. Along the same lines, the electrolaryngographic results of the present study (elevated fundamental frequency and a lower time for the closed period of the vocal folds) can be assumed to be precursors of rigidity.

Similarly to other studies that focused on finding sensitive measures of bradykinesia in early Parkinson's disease (Watts et al., 1991), future studies can help in the early diagnosis of the disease by isolating correlates of speech with the neurological symptoms of the disease. Because instrumental studies (acoustic or physiological) can effectively isolate and quantitatively measure individual variables of speech and voice, they might be useful in finding correlates of bradykinesia in running speech. Other studies can investigate if volitional non speech or speech movements (excluding running speech) of the articulators can correlate with running speech. Along the same lines, Solomon et al. (1995) employed subjects with mild Parkinson's disease (Hoehn and Yahr stages I-II) and found that tongue weakness is associated with a greater number of articulatory errors. Gender differences may also show gender specific abnormalities in speech and voice as has been suggested by other researchers (Gamboa et al., 1997; Hertrich & Ackermann, 1995; Holmes et al., 2000; Kent et al., 1994).

In the progression of hypokinetic dysarthria, increased rigidity in the speech and voice musculature expressed perceptually as monopitch,

monoloudness, reduced stress, short rushes of speech, variability in rate, and imprecise consonants will take place (Chenery et al., 1988; Darley et al., 1969a, 1969b, 1975; Logemann et al., 1978; Ludlow & Bassich, 1984). Instrumentally, an increased fundamental frequency and standard deviation of fundamental frequency (measures of long-term phonatory instability), jitter and shimmer, and a reduced range of fundamental frequency are listed in the literature (Canter, 1963; Gamboa et al., 1997; Hertrich & Ackermann, 1995; Kent et al., 1994; Ludlow & Bassich, 1983, 1984; Ludlow et al., 1983; Ramig et al., 1988; Zwirner & Barnes, 1992; Zwirner et al., 1991). Perceptually determined impairment in vocal loudness that accompanies the abnormalities in pitch (Duffy, 1995; Gentil & Pollak, 1995; Ludlow & Bassich, 1984; Schulz & Grant, 2000) has been expressed in intensity abnormalities in instrumental studies (Canter, 1965a; Ludlow & Bassich, 1983, 1984). Also, electromyographic evidence showed an increased activity of the thyroarytenoid muscle that was exhibited as reduced loudness (Baker et al., 1998) and breathiness (Gallena et al., 2001), high frequency discharges of the posterior cricoarytenoid and interarytenoid muscles (Guidi et al., 1981), and abnormalities in the function of the intrinsic laryngeal musculature (Hirose et al., 1988). This activity causes a slower opening of the vocal folds, a longer open phase, and stiffness of the vocal folds (Gerratt et al., 1987; Hanson et al., 1983). During the disease process, perceptually determined articulatory imprecision described as incomplete closure for stops and insufficient constriction for fricatives (Canter, 1965b; Chenery et al., 1988; Darley et al., 1969a, 1969b, 1975; Logemann et al., 1978; Logemann & Fisher, 1981; Zwirner & Barnes, 1992) was proven by the acoustic analysis (Ackermann & Ziegler, 1991; Kent & Rosenbek, 1982; Weismer, 1984a). Again, rigidity of the articulators and a reduced range of movement (hypokinesia) are responsible for

the production of imprecise consonants (Gentil & Pollak, 1995) and the perception of fast rate in Parkinson's disease (Kent & Rosenbek, 1982; Tjaden, 2000).

As the only study that measured voice in the progression of Parkinson's disease, Holmes et al. (2000) supported the notion that progression in severity of phonatory features is accompanied by the progression of the disease. Increased clinical disability and severity of dysarthria has also been suggested to increase with the mean fundamental frequency (Metter & Hanson, 1986). The results of the current study support the above findings by showing that no differences exist between the Parkinsonian and control groups and that variables such as mean speaking fundamental frequency, standard deviation of fundamental frequency, jitter, and shimmer are not significant in patients with early Parkinson's disease.

In theory, if the depletion of dopamine create slowness of movement in the articulators as signs of hypokinetic dysarthria, its replenishment with levodopa will ameliorate the speech symptomatology. One researcher has pointed out the importance of neurotransmitters to speech and voice production in neurological disorders (Critchley, 1981). In the beginning of Parkinson's disease, the within group results of the present study showed an improvement in the subsections of tongue and lips of the Frenchay Dysarthria Assessment. In the voice subsections however, this improvement was not so evident.

In the literature, there is no general agreement about the favourable effects of medication on hypokinetic dysarthria. While most of the studies show a beneficial effect of levodopa on speech (Cahill et al., 1998; Gallena et al., 2001; Leanderson et al., 1971; Metter & Hanson, 1986; Nakano et al., 1973; Wolfe et al., 1975) this did not occur on voice parameters (Daniels et al., 1996; Jiang et al., 1999). Phonation seems to be resistant to the favourable effect of

medication. The progression of Parkinson's disease and the dosage of levodopa medication were both suggested to relate to speech and voice problems, through the motor fluctuations that occur after 3-5 years of medication (Critchley, 1976; Duffy, 1995; Marsden & Parkes, 1976). As has been stated throughout the present study the inclusion of Parkinsonian subjects in different neurological stages and disease duration may account for these inconsistencies. The present study showed that levodopa had a beneficial effect on the areas of tongue and lips but its effect on voice remains unclear. More research is needed to confirm this finding.

Generalisation of the findings of the present study should take place with caution due to limitations inherent to its development. A main limitation is that the present study was based on findings of studies that used English speaking samples. Hypokinetic dysarthria in Greek speaking populations may show different patterns due to linguistic differences. In addition, the lack of norms regarding fundamental frequency, fundamental frequency range and intensity in normal voice production in Greek may be a further complicating factor. The small sample of the present study is certainly a restricting factor regarding the generalisation of the findings.

Moreover, methodological issues such as the use of some tools that had to be either translated in another language or constructed may be also limiting factors to the application of the results. The lack of any dysarthria assessment tool in the Greek language was a complicating factor to be overcome. Even though, the use of the translated Frenchay dysarthria assessment (FDA) was justified compared to other dysarthria tests, stages such as translation and back translation were omitted. Furthermore, piloting of the tools used in this study was also omitted.

In addition, some of the reported findings of the present study (slower movement of the articulators) are based on the maximum performance subtests of the FDA. Inherent weaknesses of such tasks exist and have been reported in the literature. Also, the use of a word list for intelligibility assessment (multiple choice testing) has its own limitations as well. It excludes factors such as situation and context and it is based on reading on the part of the subjects.

Finally, paralinguistic aspects such as mood (depression) were not assessed in the present study. Even though, there is a scarcity of studies that measure the effect of depression on voice in Parkinson's disease, the different nature of the present study (the subjects have been just diagnosed with Parkinson's disease) compared to the other studies, could justify its assessment.

The strength of the present study lies in the fact that it looks at speech-voice characteristics at the very beginning of Parkinson's disease. Dysarthria assessment at this very early stage of the disease is important in order to clarify its characteristics, excluding confounding factors (disease duration, neurological stage, and medication). The sample of this study, although small, was homogeneous in the sense that all subjects were just diagnosed with Parkinson's disease and have not been medicated. In this stage of the disease, a thorough speech assessment may help in finding clinical markers of the disease and in facilitating neurological diagnosis, especially in Parkinson's disease where the rate of error in the neurological diagnosis is high (20-25%). It was the intention of the present study to examine speech and voice variables that can mark the appearance of Parkinson's disease. An important finding is that the identification of early indicators of the dysarthria in Parkinson's disease needs to take into consideration not the phonatory characteristics of the disease

but the non-speech characteristics such as the movements of the tongue and lips.

In addition, this study intended to examine the effect of medication on speech-voice characteristics at the beginning of Parkinson's disease. Again, the sample of this study, although small, was homogeneous in the sense that all subjects had never been exposed to levodopa medication. The results showed that levodopa medication seems to affect the tongue and lips areas, while its effect on phonation needs further investigation. Even though the application of these findings on speech therapy is not direct, one can assume that since the slowness of movement of the articulators can be reversed with levodopa, the need for speech therapy at the beginning of the disease is not immediate.

Finally, even though there are inherent limitations in the use of a Greek sample (due to the lack of studies in the Greek language), the present study is the only one up to now that focused on the dysarthria of Parkinson's disease on native speakers of Greek. It constitutes a basis, from which other studies can begin, can refine its tools (translation/back translation and piloting) and assess dysarthria in the Greek speaking population in Parkinson's disease and other neurological disorders.

Future assessment with instrumental methods of non speech movements (see a review of such methods by Robin et al., 1997) and alternating motion rates (Parnell & Ammerman, 1996) may be able to add to the findings of the present study. An intelligibility assessment based on specific phonetic contrasts that require primarily tongue elevation may be tailored to relate the findings of the present study (slower movement of tongue and lips) to speech. For example, velar-null contrasts, stop-fricative contrasts, and voicing with velar involvement may show differences in patients being in the beginning of Parkinson's disease.

Logemann and Fisher (1981) report the high incidence of velars in phonetic errors in Parkinsonian subjects with variable disease duration.

In general, since there is a reported variability in Parkinsonian subjects in speech/voice (Duffy, 1995; Kent et al., 1994) and a difficulty inherent to the structure of the health system for large group studies, other research strategies may prove to be equally important. For example, research designs examining in depth the individual data of a small number of subjects may lay the groundwork for future research that will use larger groups of subjects by identifying variables that can be manipulated and by generating hypotheses that need to be tested (Schiavetti & Metz, 1997). Multiple measurements of speech/voice under different speaking tasks (time-series design) may show details that statistical comparisons of the averages may not be able to yield.

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APPENDICES

Appendix A

Definitions of terms used in the study

Agnosia: It is the inability to recognise or attach meaning to sensory information, although the physiologic receptor mechanism is intact; usually associated with a central nervous system disorder.

Agonist: 1. The muscle directly engaged in contraction as distinguished from muscles that have to relax at the same time; thus, in bending the elbow, the biceps brachii is the agonist and the triceps the antagonist. 2. In pharmacology, a drug that binds to the receptor and stimulates the receptor's function. Drugs that mimic the body's own regulatory function are called agonists.

Alternating loudness: It is expressed as alternating changes in loudness.

Alternating motion rates: Alternate repetitive movements of tongue.

Amplitude fluctuation ratio: It is a measure of the mean phottoglottography amplitude fluctuation divided by the mean phottoglottography and multiplied by 1,000.

Autosomal recessive forms of Parkinson's disease: In general, individuals with autosomal recessive forms are genetically inherited to develop Parkinson's disease.

Babinski sign: The existence of Babinski reflex in people more that 2 years old denotes some kind of incoordination, weakness and difficulty with muscle control. The Babinski reflex is the reflex of the great toe when it flexes to the top.

Basal ganglia: They involve masses of cells that are located deep in each cerebral hemisphere. They involve the caudate nucleus, putamen and globus pallidus.

Bradykinesia: Slowness of movement.

Brain stem: Portion of the brain that connects the cerebral hemispheres with the spinal cord. Neuroanatomically, it is divided to midbrain, pons and medulla oblongata.

Breathy voice: The mode of vibration of the vocal folds is inefficient and it is accompanied by slight audible friction. The muscular effort in breathy voice is low, resulting in somewhat open glottis along most of its length, so the vocal folds never meet on the mid-line. Breathiness is characterised by minimal adductive tension and weak medial compression, just sufficient to allow aerodynamic forces in the comparatively large volume of transglottal airflow to superimpose on the outflowing air.

Contracture: It is an abnormal shortening of muscle tissue, rendering the muscle highly resistant to passive stretching.

Coronary heart disease: Disease that affects the heart muscle and the blood vessels and results in heart attack due to blockage of the coronary arteries that supply blood to the heart.

Encephalitis lethargica: It is an influenza epidemic that occurred between 1914-1930 and created symptoms like bradykinesia and chorea. The survivors of this epidemic produced a form of Parkinsonism a few months to 25 years after the recovery of the disease.

Eosinophillic cytoplasmic inclusions (Lewy bodies): There are coarse, round granules of uniform size that are found in the cytoplasm of the cell.

Executive function: It is defined as high-order cognitive tasks such as planning and goal directed behaviour. More specifically, it is the ability to maintain an appropriate problem-solving set for attainment of a future goal.

Festinating gait: It is an involuntary tendency to take short accelerating steps in walking.

Harsh voice: The predominant characteristic of harsh voice is the aperiodicity of the fundamental frequency (aperiodic mode of vibration of the vocal folds or irregularity of vocal fold vibration), which is heard as a component of auditory quality rather than of auditory pitch. The physiological correlate of harshness is laryngeal tension or excessive approximation of the vocal folds. The exaggerated laryngeal tension in harsh voice is a combination of extreme adductive tension and extreme medial compression, brought about by over-contraction of the muscles.

Heredodegenerative Parkinsonism: It is the Parkinsonism that it is under genetic influence.

Hoarse voice: Laver (1980) prefers the name harsh whispery voice. This type of voice is a compound phonation in which harshness boosts adductive tension and medial compression to an extreme degree, narrowing the glottal aperture and resulting in an audible whisper rising in amplitude until the gap is completely closed. Physiologically, the whisper component is maintained by a much greater effort on the part of the lateral cricoarytenoid muscles to keep the arytenoid triangle open against the vigorous attempt by the arytenoid muscles to close it. The interaction of harshness and whisper is primarily with the voice component rather than with each other in the particular respect of aperiodicity.

Hypernasality: Excessive nasal voice when the air is resonated by the nasal cavities.

Hyponasality: Denasal voice.

Imprecise consonants: Consonant sounds lack precision. They show slurring, inadequate sharpness, distortions, and lack of crispness. There is clumsiness in going from one consonant sound to another.

Incidence: It is the number of new cases of a disorder first developed or diagnosed during a specific time interval.

Irregular articulatory breakdown: Intermittent non systematic breakdown accuracy of articulation.

Jitter: Variations of fundamental frequency in successive glottal pulses (Heiberger & Horii, 1982). Jitter measurements are concerned with how much a given period differs from the period that immediately follows it (short-term variation), and not how much it differs from a cycle at the other end of the utterance (Baken, 1997).

Levodopa: It is the direct metabolic precursor of dopamine, it acts as a neurotransmitter in the brain and it restores the low cerebral dopamine levels in Parkinson's disease (Sinemet, Sinemet CR, Madopar).

Locus coeruleus: An area with neurons that is located in the lower brain in the pons.

Mesenchephalon: Midbrain, the upper part of the brain stem.

Monoloudness: Voice shows monotony of loudness. It lacks normal variations in loudness (Darley et al., 1969a).

Monopitch: Voice is characterised by a monotone. It lacks normal pitch and inflectional changes and it tends to stay at one pitch level.

MPTP induced Parkinsonism: It is a form of Parkinsonism that was developed in young people when they were used a synthetic drug that was contaminated with the neurotoxin MPTP. MPTP-induced Parkinsonism mimics the symptoms of idiopathic Parkinson's disease.

Non propositional speech: It indicates speech produced for other reasons than the transmission of ideas and involves the automatic speech.

Oculogyric crises: Refers to crises where the movement of the eye occurs in the anterioposterior axis.

Percent jitter: It is defined as the average jitter in milliseconds divided by the average period in milliseconds times 100 (% in milliseconds).

Percent shimmer: It is defined as the percentage of average differences of peak amplitudes among successive glottal pulses relative to the average peak amplitude of the phonation (Heiberger & Horii, 1982).

Phonotactic patterning: It is defined as the sequential arrangements of phonological units which occur in a language (what counts as a phonologically well-formed word). For example, the consonant cluster /str/ is not used in the Greek language in the final position of a word.

Pigmented neurons: Coloured neurons.

Pitch breaks: Pitch of voice shows sudden and uncontrolled variation (falsetto breaks).

Pitch level: It refers to the voice that is judged to exceed the range of acceptable pitch for age and/or sex, being either too low or too high. Inappropriate pitch level is typically the hallmark of puberphonia, persistent glottal fry, and lack of pitch variability (high or low and a wide or narrow range). In neurological disorders the latter may affect the ability to control pitch and it may result in a monotone voice (Parkinson's disease). Inappropriate pitch level is a deviation from the neutral setting in that moderate longitudinal tension, adductive tension, and medial compression (inadequate medial compression in case of Parkinson's disease) result in a regular periodic vocal fold production.

Prevalence: It refers to the total number of persons with a disorder within a given population at a fixed point in time.

Progressive Supranuclear Palsy or Supranuclear Gaze Palsy: It is a neurological degenerative disease that destroys nerve cells of primarily the pons and the midbrain. Its symptoms include difficulty with voluntary eye movement, problems in walking, frequent falls or stiff slow movements of the arms and legs.

Propositional speech: Jonas (1981) describes it as the speech that is produced by a speaker who intends to transmit ideas.

Propulsion: It is a tendency to fall forward in walking.

Rate: Abnormally rapid or slow perceivable rate.

Reduced stress: Minimal amount of emphasis given a syllable in a word; such syllables are short, often low in pitch, and indefinite in quality.

Red nucleus, nucleus basalis and raphe nuclei: Cell bodies that are located in the midbrain.

Retropulsion: It is defined as an abnormal gait in which the body is bent backward.

Shimmer: It is defined as variations of peak amplitude in successive glottal pulses (Heiberger & Horii, 1982).

Shy-Drager syndrome: Progressive neurological disorder that affects the motor components of the autonomic and somatic portions of the central nervous system.

Short rushes of speech: They are defined as short rushes of speech that are separated by pauses.

Strained-strangled voice: It is expressed as an effort to squeeze the voice through glottis. It is also called ventricular voice by Laver. It occurs when the ventricular folds become involved in the phonation of the true vocal folds, with the effect that the true and the ventricular folds combine to vibrate as more massive, composite elements. In order to bring the ventricular folds tot his position, a high degree of muscular tension is needed, and the effect is normally to make phonation auditorily very harsh.

Striatum: It is a part of the basal ganglia involving the structures, caudate nucleus and putamen.

Substantia nigra: It is a deeply pigmented area of the midbrain containing dopamine-producing nerve cells. Neurophysiologically, the substantia nigra is considered as a part of the basal ganglia. Neuroanatomically, they are divided to substantia nigra pars compacta (cell region of large pigmented neurons) and substantia nigra pars reticulata.

Variable rate: Excessively fast or laboriously slow rates due to (among others) neurological involvement. Persons who tend to talk very rapidly often tend to stretch vocalisation to the last bit of air they can squeeze out. The prosody of speech may be disturbed by vocal behaviour. In the vocal profile analysis protocol the inappropriate fast or slow rate of speech has been measured in severity in scalar degrees.

Ventral tegmental area: It is an area of the midbrain (upper part of brain stem) from which the axons that leaving the cells of the red nucleus decussate and course to the spinal cord.

Appendix B

Hoehn and Yahr Neurological Scale

Stage 0: No signs of disease.

Stage 1: **Unilateral disease.**

The disease in this stage is characterised by mild resting tremor, rigidity, bradykinesia, dysarthria, trunk tilt, fine motor incoordination, and facial immobility. These symptoms are noticeable but not disabling. Symptoms often present in unilateral or hemiparetic fashion.

Stage 2: **Bilateral disease, without impairment of balance.**

The person is mildly disabled as symptoms appear bilaterally and standing posture becomes stooped. Gait is a shuffle. Fatigue, bradykinesia, and weakness impair home and work activities. The wrist assumes a slightly dorsiflexed position. The flexed metacarpophalangeal and distal and extended proximal interphalangeal joints characterise the hand deformity. A pattern of truncal and limb contracture is noted.

Stage 3: **Mild to moderate bilateral disease.**

Moderate disability involves a festinating gait. Retropulsion initially and propulsion later interfere with stopping, starting, turning, and stepping backward. Self-care activities are tediously performed and often require attendant help. Falls become a real threat to the person's safety.

Stage 4: **Severe disability.**

Marked rigidity, akinesis, and poor standing balance are severely disabling, as is fine motor incoordination. Thus, safe independent

ambulation is confined to the home at best and self-care skills require assistance. Contractures are increasingly more refractory to conventional stretching exercises. Tremor, interestingly, is less pronounced.

Stage 5: **Wheelchair bound or bedridden**.

This stage presents complete dependency and serious worsening of all preceding patterns of musculoskeletal disability. Aspiration, pneumonitis, weight loss, malnutrition, dehydration, and fecal impaction often necessitate a gastrostomy or nasogastric feeding tube. Many people do not reach this grave level of disability owing to fatal complications in earlier stages.

Appendix C

Patient_____

Examiner_____

Date_____

"MINI-MENTAL STATE"

Maximum Score	Score	
		ORIENTATION
5	( )	What is the (year) (season) (date) (day) (month)?
5	( )	Where are we: (state) (county) (town) (hospital) (floor).
		REGISTRATION
3	( )	Name 3 objects: 1 second to say each. Then ask the patient all after you have said them. Then repeat them until he learns all Count trials and record.
		Trials
		ATTENTION AND CALCULATION
5	( )	Serial 7's. 1 point for each correct. Stop after 5 answers. Alternatively spell "world" backwords.
		RECALL
3	( )	Ask for the 3 objects repeated above. Give 1 point for each correct.
		LANGUAGE
9	( )	Name a pencil, and watch (2 points). Repeat the following "No ifs, ands or buts." (1 point) Follow a 3-stage command: "Take a paper in your right hand, fold it in half, and put it on the floor" (3 points) Read and obey the following: CLOSE YOUR EYES (1 point) Write a sentence (1 point) Copy design (1 point)
_____		Total score

ASSESS level of consciousness along a continuum

Alert Drowsy Stupor Coma

Όνομα ασθενούς _____
Εξεταστής _____
Ημερομηνία _____

"MINI MENTAL STATE"

Maximum

Score Score

ΠΡΟΣΑΝΑΤΟΛΙΣΜΟΣ

5 ( ) Ποια είναι (η χρονιά) (η εποχή) (η ημερομηνία) (η μέρα) και (ο μήνας);
5 ( ) Πού βρισκόμαστε: (χώρα), (νομός) (πόλη) (μέρος –σπίτι), (όροφος)

ΑΝΑΓΝΩΡΙΣΗ – ΜΝΗΜΗ

3 ( ) Ονομάστε 3 πράγματα:1 δευτερόλεπτο για το καθένα. Μετά ρωτήστε τον ασθενή και για τα τρία αφού τα έχετε ονομάσει όλα. Επαναλάβετε μέχρι να τα μάθει και τα τρία. Μετρήστε προσπάθειες και γράψτε τις
Προσπάθειες :

ΠΡΟΣΟΧΗ – ΥΠΟΛΟΓΙΣΜΟΣ

5 ( ) Μετράτε ανά 7. Ένα βαθμό για κάθε σωστή απάντηση.
Σταματήστε μετά από 5 απαντήσεις. Εναλλακτικά
να πεί την λέξη –κόσμος από την ανάποδη

ΜΝΗΜΗ

3 ( ) Ρωτήστε για τα 3 πράγματα στην παραπάνω άσκηση.
Βάλτε ένα βαθμό για κάθε σωστή απάντηση.

ΓΛΩΣΣΑ

9 ( ) Ονομάστε ένα μολύβι, και ένα ρολόι (2 βαθμοί)
Επαναλάβετε το παρακάτω "Σήμερα έχει πολύ ζέστη έξω" (1 βαθμός)
Ακολουθήστε μία εντολή 3 σταδίων:
"Πάρτε ένα χαρτί στο δεξί σας χέρι, διπλώστε το στην μέση και βάλτε το στο πάτωμα" (3 βαθμοί)
Διαβάστε και ακολουθήστε το παρακάτω:
ΚΛΕΙΣΤΕ ΤΑ ΜΑΤΙΑ ΣΑΣ (1 βαθμός)
Γράψτε μία πρόταση (1 βαθμός)
Αντιγράψτε το σχέδιο (1 βαθμός)

_____ Τελικό σκορ

ΣΥΝΕΙΔΗΣΗ

Αξιολογήστε το επίπεδο συνείδησης πάνω σε μία συνεχή γραμμή

Σε εγρήγορση Ζαλισμένος/η Σε Λήθαργο Σε Κώμα

Appendix D

ΕΜΠΙΣΤΕΥΤΙΚΟ

Ημερομηνία: _____

Αριθμός Ασθενούς:_____

Ώρα Έναρξης: _____

ΙΣΤΟΡΙΚΟ ΓΙΑ ΕΚΤΙΜΗΣΗ ΟΜΙΛΙΑΣ

Όνομα Ασθενούς:_____Ημερ/νία Γέννησης-Ηλικία: _____

Επάγγελμα: _____Τόπος Γέννησης: _____

Προσωπική Κατάσταση: _____ Εκπαίδευση: _____

Οικογενειακή Κατάσταση: _____Φύλο: _____

Διεύθυνση: _____

Ημερ. Διάγνωσης: _____ Τηλέφωνο:_____

	Ναι	Όχι
1. Υπάρχει κάποιο ιστορικό προβλημάτων λόγου ή ομιλίας στην παιδική σας ηλικία;	_____	_____
2. Ακολουθήσατε κάποια θεραπεία για αυτό;	_____	_____
3. Εχετε κάποια δυσκολία με την ακοή σας;	_____	_____
4. Αν ναι, πότε αυτή εμφανίστηκε και για πόσο διήρκεσε;	_____	
_____		
5. Εχετε κάποια προβλήματα με την όρασή σας;	_____	_____
6. Αν ναι, τί είδους προβλήματα;_____		
7. Μπορείτε και διαβάζετε παρόλα αυτά τα προβλήματα;	_____	_____
8. Αν όχι, γιατί συμβαίνει αυτό;_____		

Εμφάνιση και Διάρκεια

9. Έχετε κάποια δυσκολία με την ομιλία σας;	_____	_____
10. Αν όχι, έχει κάποιος άλλος αναφέρει για καμμία αλλαγή ή πρόβλημα στην ομιλία σας;_____		
11. Πότε εμφανίστηκε το πρόβλημα στην ομιλία σας;_____		
12. Εμφανίστηκε ξαφνικά ή σταδιακά;_____		
13. Ποιός το αντιλήφθηκε πρώτος, εσείς ή κάποιος άλλος;_____		

ΕΜΠΙΣΤΕΥΤΙΚΟ

14. Αναπτύξατε κάποιες άλλες δυσκολίες όταν το πρόβλημα στην ομιλία σας εμφανίστηκε;_____
15. Αυτές εμφανιστήκαν πριν ή μετά από το πρόβλημα ομιλίας;_____
16. Έχει αλλάξει το πρόβλημα ομιλίας;

    Καλύτερα _____ Χειρότερα _____

    Σταθερό _____

    Μεταβάλλεται προς (περιγραφή) _____

	Ναι	Όχι
17. Έχει επανέλθει ποτέ η ομιλία σας στο κανονικό;	_____	_____
18. Αν ναι, πότε και για πόσο διάστημα;_____		
19. Έχετε κάποια δυσκολία στην μάσηση;	_____	_____
20. Μήπως έχετε παρατηρήσει να ΜΗΝ υπάρχει συμμετρία στο πρόσωπό σας;	_____	_____
21. Είναι δύσκολο να μετακινείτε το φαγητό γύρω μέσα στο στόμα σας;	_____	_____
22. Γιατί;_____		
23. Μήπως το φαγητό κολλάει στα μάγουλά σας ή στο πάνω μέρος του στόματός σας;	_____	_____
24. Χρειάζεται να το βγάζετε με το δάκτυλό σας ή με το πηρούνι;	_____	_____
25. Έχετε δυσκολία να μετακινείτε το φαγητό στο πίσω μέρος του στόματός σας, έτσι ώστε να αρχίσετε να καταπίνετε;_____		_____
26. Έχετε δυσκολία στην κατάποση;	_____	_____
27. Στα φαγητά ή στα υγρά; _____		
28. 'Εχετε δυσκολία όταν αρχίζετετε να καταπίνετε;	_____	_____
29. Σας πέφτει έξω από το στόμα σας φαγητό ή υγρό;	_____	_____
30. Κατά την διάρκεια της κατάποσης έχει ποτέ εισχωρήσει ή εξέλθει από την μύτη σας φαγητό ή υγρό;	_____	_____
31. Κατεβαίνει ποτέ φαγητό ή υγρό πριν να καταπιείτε, έτσι ώστε να σας προκαλέσει βήχα ή και πνίξιμο;	_____	_____
32. Μήπως σας έρχεται να βγάλετε το φαγητό καθώς καταπίνετε;	_____	_____
33. Μήπως βήχετε λίγο μετά αφού έχετε καταπιεί;	_____	_____

ΕΜΠΙΣΤΕΥΤΙΚΟ

34. Χρειάστηκε να αλλάξετε την δίαιτά σας λόγω αυτών των προβλημάτων; _____ _____

35. Έχετε χάσει βάρος; _____ _____

	Ναι	Όχι
36. Έχετε παρατηρήσει καμμία αλλαγή στην συναισθηματική σας κατάσταση;	_____	_____
37. Κλαίτε ή γελάτε πιο εύκολα ή πιο δύσκολα από το παρελθόν;	_____	_____
38. Παίρνετε κάποια φάρμακο που φαίνεται ότι επηρεάζει την ομιλία σας;	_____	_____

39. Αν ναι ποια; _____

Η αντίληψη του ασθενούς για το πρόβλημα

40. Πώς ακουγόταν η ομιλία/φωνή σας όταν το πρόβλημα άρχισε;_____

_____

41. Περιγράψτε μου την τωρινή δυσκολία στην ομιλία/φωνή σας

πιο γρήγορη	_____	ή	_____	πιο αργή
πιο δυνατή	_____	ή	_____	πιο σιγανή
πιο ακριβής	_____	ή	_____	λιγότερο ακριβής

42. Χρειάζεται φυσική προσπάθεια για να μιλήσετε; _____ _____

43. Έχετε παρατηρήσει κάποια αλλαγή στήν εμφάνιση ή στην αφή του προσώπου σας και του στόματός σας; _____ _____

44. Αν ναι, τί είδους αλλαγή;_____

Συνέπειες του προβλήματος

45. Οι άνθρωποι έχουν πρόβλημα να σας καταλαβαίνουν; _____ _____

46. Αν ναι, σε ποιες περιπτώσεις;_____

47. Τί κάνετε όταν αυτό συμβαίνει;_____

48. Χρειάζετε ποτέ να γράψετε για να σας καταλάβουν; _____ _____

49. Αν ναι, το πρόβλημα στην ομιλία σας έχει επηρεάσει την δουλειά σας; _____ _____

50. Αν ναι, σας έχει σταματήσει από το να κάνετε οτιδήποτε;_____ _____

ΕΜΠΙΣΤΕΥΤΙΚΟ

Αντιμετώπιση

51. Τί έχετε κάνει για να αντισταθμίσετε το πρόβλημα στην ομιλία σας;_____

_____

52. Νομίζετε ότι χρειάζεστε βοήθεια με την ομιλία σας τώρα;_____

_____

Επίγνωση της διάγνωσης και της πρόγνωσης

53. Τί σας έχουν πει για την αιτία αυτού του προβλήματος;_____

_____

54. Όσον αφορά την διάγνωση, γνωρίζετε τί θα επακολουθήσει;_____

_____

Άλλες παρατηρήσεις βασισμένες στο Ιστορικό του ασθενούς

Κάπνισμα _____

Ποτό _____

Άλλα _____

_____

_____

_____

_____

Φαρμακευτική αγωγή πριν τη διάγνωση_____

_____

_____

_____

_____

_____

_____

_____

_____

_____

_____

_____

Ώρα παύσης: _____

CONFIDENTIAL

Date: _____

Subject Number: _____

Time Begun: _____

HISTORY FORM FOR MOTOR SPEECH EXAMINATION

Patient's name:_____ Date of Birth/Age: _____

Occupation: _____ Place of Birth: _____

Marital Status: _____ Education: _____

Family Status: _____ Sex: _____

Address: _____

Date of Diagnosis_____ Telephone:_____

	Yes	No
1. Was there any history of childhood speech, and language deficit?	____	____
2. Have you received any treatment for that?	____	____
3. Do you have any difficulty with your hearing?	____	____
4. If yes, when did that happen and for how long? _____		
_____		
5. Do you have any problems with your vision?	____	____
6. If yes what is it? _____		
7. Can you read despite these problems?	____	____
8. If no, why is that? _____		

Onset and Course

9. Do you have any difficulty with your speech?	____	____
10. If not, has anyone else commented on a change or problem with your speech? _____		
11. When did the speech problem begin?_____		
12. Did it begin suddenly or gradually? _____		
13. Who noticed it first, you or someone else? _____		

14. Did you develop any other difficulties when your speech?
problem began? _____
15. Were there before or after the speech problem? _____
16. Has the speech problem changed?
Better _____ Worse _____
Stable _____ Fluctuating _____
Better-then-Stable _____

	Yes	No
17. Has your speech ever returned to normal?	____	____
18. If so, when and for how long? _____		
19. Have you had any difficulty with chewing?	____	____
20. Drooling?	____	____
21. Is it difficult to move food around in your mouth?	____	____
22. Why? _____		
23. Does food get stuck in your cheeks or on the roof of your mouth?	____	____
24. Do you have to remove it with your finger or fork?	____	____
25. Do you have trouble moving food back in your mouth to get a swallow started?	____	____
26. Do you have trouble with swallowing?	____	____
27. Food or liquid? _____		
28. Do you have trouble getting a swallow started?	____	____
29. Do you loose food or liquid out of your mouth?	____	____
30. Does food or liquid ever go into or out of your nose when you swallow?	____	____
31. Does food or liquid go down before you swallow and cause coughing or choking?	____	____
32. Do you gag or choke when swallowing?	____	____
33. Do you choke or cough after completing a swallow?	____	____
34. Have you had to modify your diet because of these problems?	____	____
35. Have you lost weight?	____	____
36. Have you had any change in your emot. expression?	____	____

37. Do you cry or laugh more easily or less easily than in the past? ____ ____

38. Are you taking any medications that seem to affect your speech? ____ ____

39. State some of them: _____

The patient's perception of deficit

40. What did your speech/voice sound like when the problem began? _____

41. Describe your current speech/voice difficulty:

Faster	vs	Slower
_____		____
Louder	vs	Softer
_____		____
Precise	vs	Less precise
_____		____

	Yes	No
42. Is speaking effortful?	____	____
43. Have you noticed any change in the appearance or feeling in your face or mouth?	____	____

44. If yes, what? _____

Consequences of the disorder

45. Do people ever have trouble understanding you?	____	____
46. If yes, in which cases? _____		
47. What do you do if that happens? _____		
48. Do you ever have to write to make yourself understood?	____	____
49. If yes, has your speech problem affected your work?	____	____
50. If yes, does it prevent you from doing anything?	____	____

Management

51. What have you done to compensate for your speech difficulty? _____

52. Do you think you need help with your speech now?_____

_____

Awareness of diagnosis and prognosis

53. What have you been told is the cause of this problem?

_____

54. In view of this diagnosis, what is going to happen?

_____

Other Remarks based on History

Smoking_____

Drinking_____

Other_____

_____

_____

_____

Medication before diagnosis_____

_____

_____

_____

_____

_____

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Time Ended: _____

Appendix E

Phonetic contrasts in the Greek intelligibility list of words

1.	Bad[13]	Bed	Bat	Pad
	Πήζω	**Παίζω**	**Πίσω**	**Μπήζω**
IPA.	∀πιζο	∀πΕζο	∀πισο	∀βιζο
	∀πιζο-∀πΕζο=	high-mid vowel contrast (vowel duration contrast in Kent's list)		
	∀πιζο-∀πισο=	voicing contrast medial consonant (voicing contrast final consonant in Kent's list)		
	∀πιζο-∀βιζο=	voicing contrast initial consonant		
2.	Sip	Ship	Tip	Zip
	Χώμα	**Σώμα**	**Κώμα**	**Γόμα**
IPA.	∀ξομ6	∀σομ6	∀κομ6	∀Γομ6
	∀ξομ6-∀σομ6=	alveolar-velar place contrast (alveolar-palatal place contrast in Kent's list)		
	∀ξομ6-∀κομ6=	stop-fricative contrast		
	∀ξομ6-∀Γομ6=	voicing contrast initial consonant		
3.	Spit	Pit	Sit	It
	Πλήθος	**Πίθος**	**Λίθος**	**Ήθος**
IPA.	∀πλιΤοσ	∀πιΤοσ	∀λιΤοσ	
	∀ιΤοσ			
	∀πλιΤοσ-∀πιΤοσ=	initial cluster-initial singleton contrast		
	∀πλιΤοσ-∀λιΤοσ=	initial cluster-initial singleton contrast		
	∀πλιΤοσ-∀ιΤοσ=	initial consonant-null contrast		
4.	Knot	Dot	Nod	Nut
	Νότα	**Ρότα**	**Νώντα**	**Νάτα**
IPA.	∀νοτ6	∀4οτ6	∀νονδ6	
	∀ν6τ6			
	∀νοτ6-∀4οτ6=	nasal-glide contrast (stop-nasal contrast in Kent's list)		
	∀νοτ6-∀νονδ6=	voicing contrast medial consonants (voicing contrast final consonant in Kent's list)		
	∀νοτ6-∀ν6τ6 =	mid-low vowel contrast (vowel duration contrast in Kent's list)		
5.	Sigh	Shy	Tie	Thigh
	Ποσό	**Ποθώ**	**Ποτό**	**Ποσού**
IPA.	πο∀σο	πο∀Το	πο∀το	πο∀συ
	πο∀σο-πο∀Το =	alveolar-dental place contrast (alveolar-palatal or postalveolar place contrast in Kent's list)		
	πο∀σο-πο∀το=	stop-fricative contrast		
	πο∀σο-πο∀συ=	high-mid vowel contrast (other fricative contrast in Kent's list)		
6.	Sheet	Seat	Feet	Eat
	Χήτα	**Σήτα**	**Θήτα**	**Ήτα**
IPA.	∀ξιτ6	∀σιτ6	∀Τιτ6	∀ιτ6
	∀ξιτ6-∀σιτ6=	alveolar-velar place contrast (alveolar-palatal or postalveolar place contrast in Kent's list)		
	∀ξιτ6-∀Τιτ6=	Other fricative contrast		
	∀ξιτ6-∀ιτ6=	initial consonant-null contrast		
7.	Sticks	Six	Ticks	Stick
	Κρίμα	**Κύμα**	**Ρήμα**	**Κρίμας**
IPA.	∀κ4ιμ6	∀κιμ6	∀4ιμ6	∀κ4ιμ6σ
	∀κ4ιμ6-∀κιμ6=	initial cluster-initial singleton contrast		

[13] The first row of each number (1, 2, etc.) represents Kent's et al. (1989) words and the second row the corresponding Greek words.

	∀κ4ιμ6-∀4ιμ6=	initial cluster-initial singleton contrast		
	∀κ4ιμ6-∀κ4ιμ6σ=	final cluster-final singleton contrast		
8.	Knew	Know	Knee	Gnaw
	Του	**Το**	**Τι**	**Τα**
IPA.	τυ	το	τι	τ6
	τυ-το=	high-mid vowel contrast (high-low vowel contrast in Kent's list)		
	τυ-τι=	front-back vowel contrast (front-back vowel contrast in Kent's list)		
	τυ-τ6=	high-low vowel contrast		
9.	Leak	Lick	League	Reek
	Λύσσα	**Λούσα**	**Λίζα**	**Ρίσα**
IPA.	∀λισ6	∀λυσ6	∀λιζ6	∀4ισ6
	∀λισ6-∀λυσ6=	front-back vowel contrast (vowel duration contrast in Kent's list)		
	∀λισ6-∀λιζ6=	voicing contrast medial consonant (voicing contrast final consonant in Kent's list)		
	∀λισ6-∀4ισ6=	/r/-/l/ glide-lateral approximant contrast (/r/-/l/ contrast in Kent's list)		
10.	Chair	Share	Tear	Air
	Τσίμα	**Σήμα**	**Τίμα**	**Ήμα**
IPA.	∀τσιμ6	∀σιμ6	∀τιμ6	∀ιμ6
	∀τσιμ6-∀σιμ6=	fricative-affricate contrast		
	∀τσιμ6-∀τιμ6=	stop-affricate contrast		
	∀τσιμ6-∀ιμ6 =	initial consonant-null contrast		
11.	Nice	Knife	Night	Dice
	Μάσα	**Μάζα**	**Μάτα**	**Μπάσα**
IPA.	∀μ6σ6	∀μ6ζ6	∀μ6τ6	∀β6σ6
	∀μ6σ6-∀μ6ζ6=	voiced-voiceless fricative contrast (other fricative contrast in Kent's list)		
	∀μ6σ6-∀μ6τ6=	stop-fricative contrast		
	∀μ6σ6-∀β6σ6=	stop-nasal contrast		
12.	Write	Ride	Light	White
	Ρύση	**Ρύζι**	**Λύση**	**Δύση**
IPA.	∀4ισι	∀4ιζι	∀λισι	∀Δισι
	∀4ισι-∀4ιζι=	voicing contrast medial consonants (voicing contrast final consonants in Kent's list)		
	∀4ισι-∀λισι=	/r/-/l/ glide-lateral approximant contrast (/r/-/l/ liquid) contrast in Kent's list		
	∀4ισι-∀Δισι=	glide-fricative contrast (/r/-/w/ contrast in Kent's list)		
13.	Side	Sign	Sight	Sigh
	Μάγκα	**Μάνα**	**Μάκα**	**Μα**
IPA.	∀μ6γ6	∀μ6ν6	∀μ6κ6	μ6
	∀μ6γ6-∀μ6ν6=	stop-nasal contrast		
	∀μ6γ6-∀μ6κ6=	voicing contrast medial consonant (voicing contrast final consonant in Kent's list)		
	∀μ6γ6-μ6=	disyllable-monosyllable contrast (final consonant-null contrast in Kent's list)		
14.	Pat	Bat	Pot	Pad
	Θα Σώσω	**Θα Ζώσω**	**Θα Σήσω**	**Θα Σώζω**
IPA.	Τ6 ∀σοσο	Τ6 ∀ζοσο	Τ6 ∀σισο	Τ6 ∀σοζο
	Τ6 ∀σοσο-Τ6 ∀ζοσο=	voicing contrast initial consonant		
	Τ6 ∀σοσο-Τ6 ∀σισο=	front-back and high-low vowel contrast (front-back vowel contrast in Kent's list)		
	Τ6 ∀σοσο-Τ6 ∀σοζο=	voicing contrast medial consonant (voicing contrast final consonant in Kent's list)		

15.	Hand	And	Sand	Fanned
	Χάλι	**Άλλη**	**Σάλι**	**Ζάλη**
IPA.	∀ξ6λι	∀6λι	∀σ6λι	∀ζ6λι
	∀ξ6λι-∀6λι=	initial velar-null contrast		
	∀ξ6λι-∀σ6λι=	other fricative contrast		
	∀ξ6λι-∀ζ6λι=	other fricative contrast		
16.	Ate	Hate	Aid	Fate
	Ήδη	**Γίδι**	**Ήθη**	**Φίδι**
IPA.	∀ιΔι	∀ΓιΔι	∀ιΤι	∀φιΔι
	∀ιΔι-∀ΓιΔι=	initial velar-null contrast (initial glottal-null contrast in Kent's list)		
	∀ιΔι-∀ιΤι=	voicing contrast medial consonant (voicing contrast final consonant in Kent's list)		
	∀ιΔι-∀φιΔι=	initial consonant-null contrast		
17.	Witch	Wish	Rich	Wit
	Λίτσα	**Λύσσα**	**Νίτσα**	**Λίτα**
IPA.	∀λιτσ6	∀λισ6	∀νιτσ6	∀λιτ6
	∀λιτσ6-∀λισ6=	fricative-affricate contrast		
	∀λιτσ6-∀νιτσ6=	lateral-nasal contrast (/r/-/w/ contrast in Kent's list)		
	∀λιτσ6-∀λιτ6=	stop-affricate contrast		
18.	Much	Mush	Mut	Muck
	Κάζο	**Κάντο**	**Κάνω**	**Κάρο**
IPA.	∀κ6ζο	∀κ6δο	∀κ6νο	∀κ64ο
	∀κ6ζο-∀κ6δο=	stop-fricative contrast (fricative-affricate contrast in Kent's list)		
	∀κ6ζο-∀κ6νο=	fricative-nasal contrast (stop-affricate contrast in Kent's list)		
	∀κ6ζο-∀κ64ο=	fricative-glide contrast (stop-affricate contrast in Kent's list)		
19.	Sew	Shoe	Toe	Foe
	Δένω	**Ζένω**	**Ντένω**	**Βαίνω**
IPA.	∀ΔΕνο	∀ζΕνο	∀δΕνο	∀ϖΕνο
	∀ΔΕνο-∀ζΕνο=	alveolar-dental place contrast (alveolar-palatal place contrast in Kent's list)		
	∀ΔΕνο-∀δΕνο=	stop-fricative contrast		
	∀ΔΕνο-∀ϖΕνο=	other fricative contrast		
20.	Feed	Food	Feet	Fee
	Μπεις	**Μπας**	**Μπιζ**	**Μπι**
IPA.	βισ	β6σ	βιζ	βι
	βισ-β6σ=	front-back and high-low vowel contrast (front-back vowel contrast in Kent's list)		
	βισ-βιζ=	voicing contrast final consonant		
	βισ-βι=	final consonant-null contrast		
21.	Him	Hem	Ham	Hum
	Πείρα	**Πέρα**	**Πάρα**	**Πούρα**
IPA.	∀πι46	∀πΕ46	∀π646	∀πυ46
	∀πι46-∀πΕ46=	high-mid vowel contrast (high-low vowel contrast in Kent's list)		
	∀πι46-∀π646=	high-low and front-back vowel contrast (high-low vowel contrast in Kent's list)		
	∀πι46-∀πυ46=	front-back vowel contrast		
22.	At	Hat	Fat	Add
	Είδες	**Γίδες**	**Βίδες**	**Είθες**
IPA.	∀ιΔΕσ	∀7ιΔΕσ	∀ϖιΔΕσ	
	∀ιΤΕσ			
	∀ιΔΕσ-∀7ιΔΕσ=	initial velar-null contrast (initial glottal-null contrast in Kent's list)		
	∀ιΔΕσ-∀ϖιΔΕσ=	initial consonant-null contrast		

	∀ιΔΕσ-∀ιΤΕσ=	voicing contrast medial consonant (voicing contrast final consonant in Kent's list)		
	∀7ιΔΕσ-∀ϖιΔΕσ=	other fricative contrast		
23.	Air	Hair	Fair	Are
	Ένα	**Χένα**	**Πένα**	**Άννα**
IPA.	∀Εν6	∀ξΕν6	∀πΕν6	∀6ν6
	∀Εν6-∀ξΕν6=	initial velar-null contrast (initial glottal-null contrast in Kent's list)		
	∀Εν6-∀πΕν6=	initial consonant-null contrast		
	∀Εν6-∀6ν6=	front-back and high-low vowel contrast (front-back vowel contrast in Kent's list)		
24.	Pit	Pet	Pat	Bit
	Βίρα	**Βέρα**	**Βάρα**	**Φύρα**
IPA.	∀ϖι46	∀ϖΕ46	∀ϖ646	∀φι46
	∀ϖι46-∀ϖΕ46=	high-mid vowel contrast (high-low vowel contrast in Kent's list)		
	∀ϖι46-∀ϖ646=	high-low and front-back vowel contrast (high-low vowel contrast in Kent's list)		
	∀ϖι46-∀φι46=	voicing contrast initial consonant		
25.	Read	Lead	Weed	Rrid
	Ρίμα	**Λίμα**	**Χύμα**	**Ρέμα**
IPA.	∀4ιμ6	∀λιμ6	∀ξιμ6	∀4Εμ6
	∀4ιμ6-∀λιμ6=	/r/-/l/ glide-lateral approximant contrast (/r/-/l/ liquid contrast in Kent's list)		
	∀4ιμ6-∀ξιμ6=	glide-fricative contrast (/r/-/w/ in Kent's list)		
	∀4ιμ6-∀4Εμ6=	high-mid vowel contrast (vowel duration contrast in Kent's list)		
26.	Sell	Shell	Tell	Fell
	Ζώνω	**Γόνο**	**Ντώνω**	**Σώνω**
IPA.	∀ζονο	∀Γονο	∀δονο	∀σονο
	∀ζονο-∀Γονο=	alveolar-velar place contrast (alveolar-palatal place contrast in Kent's list)		
	∀ζονο-∀δονο=	stop-fricative contrast		
	∀ζονο-∀σονο=	voiced-voiceless fricative contrast (other fricative contrast in Kent's list)		
27.	Blend	Bend	Lend	End
	Θρέμμα	**Θέμα**	**Ρέμα**	**Αίμα**
IPA.	∀Τ4Εμ6	∀ΤΕμ6	∀4Εμ6	∀Εμ6
	∀Τ4Εμ6-∀ΤΕμ6=	initial cluster-initial singleton contrast		
	∀Τ4Εμ6-∀4Εμ6=	initial cluster-initial singleton contrast		
	∀Τ4Εμ6-∀Εμ6=	initial cluster-null contrast		
28.	Shoot	Suit	Sheet	Shot
	Κοίτα	**Πίτα**	**Κούτα**	**Κότα**
IPA.	∀κιτ6	∀πιτ6	∀κυτ6	∀κοτ6
	∀κιτ6-∀πιτ6=	bilabial-velar place contrast (alveolar-palatal place contrast in Kent's list)		
	∀κιτ6-∀κυτ6=	front-back vowel contrast		
	∀κιτ6-∀κοτ6=	high-mid and front-back vowel contrast (high-low vowel contrast)		
29.	See	She	He	Tea
	Θύρα	**Φύρα**	**Χήρα**	**Τήρα**
IPA.	∀Τι46	∀φι46	∀ξι46	∀τι46
	∀Τι46-∀φι46=	labiodental-dental place contrast (alveolar-palatal place contrast in Kent's list)		
	∀Τι46-∀ξι46=	other fricative contrast		

	∀Τι46-∀τι46=	stop-fricative contrast		
30.	Slip	Sip	Lip	Sleep
	Πράσσο	**Πάσο**	**Ράσο**	**Πρόσω**
IPA.	∀π46σο	∀π6σο	∀46σο	
	∀π4οσο			
	∀π46σο-∀π6σο=	initial cluster-initial singleton contrast		
	∀π46σο-∀46σο=	initial cluster-initial singleton contrast		
	∀π46σο-∀π4οσο=	mid-low vowel contrast (vowel duration contrast in Kent's list)		
31.	Steak	Snake	Take	Sake
	Τρίμμα	**Τμήμα**	**Τίμα**	**Ρήμα**
IPA.	∀τ4ιμ6	∀τμιμ6	∀τιμ6	∀4ιμ6
	∀τ4ιμ6-∀τμιμ6=	glide-nasal in a cluster contrast (stop-nasal contrast in Kent's list)		
	∀τ4ιμ6-∀τιμ6=	initial cluster-initial singleton contrast		
	∀τ4ιμ6-∀4ιμ6=	initial cluster-initial singleton contrast		
32.	Blow	Low	Bow	Bloat
	Στρίβει	**Τρίβει**	**Στίβει**	**Στρίβεις**
IPA.	∀στ4ιϖι	∀τ4ιϖι	∀στιϖι	
	∀στ4ιϖισ			
	∀στ4ιϖι-∀τ4ιϖι=	initial cluster (3 consonants)-initial cluster (2 consonants) contrast (initial cluster-initial singleton in Kent's list)		
	∀στ4ιϖι-∀στιϖι=	initial cluster (3 consonants)-initial cluster (2 consonants) contrast (initial cluster-initial singleton in Kent's list)		
	∀στ4ιϖι-∀στ4ιϖισ=	final consonant-null contrast		
33.	Beat	Boot	Bit	Meat
	Κοίτη	**Καίτη**	**Κάτι**	**Χύτη**
IPA.	∀κιτι	∀κΕτι	∀κ6τι	∀ξιτι
	∀κιτι-∀κΕτι=	high-mid vowel contrast (front-back vowel contrast in Kent's list)		
	∀κιτι-∀κΕτι=	front-back and high-low vowel contrast (vowel duration contrast in Kent's list)		
	∀κιτι-∀ξιτι=	stop-fricative contrast (stop-nasal contrast in Kent's list)		
34.	Sin	Shin	In	Tin
	Σόλα	**Φόλα**	**Όλα**	**Τόλα**
IPA.	∀σολ6	∀φολ6	∀ολ6	∀τολ6
	∀σολ6-∀φολ6=	alveolar-labiodental place contrast (alveolar-palatal place contrast in Kent's list)		
	∀σολ6-∀ολ6=	Initial consonant-null contrast		
	∀σολ6-∀τολ6=	stop-fricative contrast		
35.	Rock	Walk	Lock	Rocks
	Ράμπα	**Ζάμπα**	**Λάμπα**	**Ράμπας**
IPA.	∀46β6	∀ζ6β6	∀λ6β6	∀46β6σ
	∀46β6-∀ζ6β6=	glide-fricative contrast (/r/-/w/ contrast in Kent's list)		
	∀46β6-∀λ6β6=	/r/-/l/ glide-lateral approximant contrast (/r/-/l/ liquid contrast in Kent's list)		
	∀46β6-∀46β6σ=	final consonant-null contrast (final cluster-final singleton contrast in Kent's list)		
36.	Geese	Goose	Guess	Gas
	Κινώ	**Κουνώ**	**Κενό**	**Κανό**
IPA.	κι∀νο	κυ∀νο	κΕ∀νο	κ6∀νο
	κι∀νο-κυ∀νο=	front-back vowel contrast		
	κι∀νο-κΕ∀νο=	high-mid vowel contrast (high-low vowel contrast in Kent's list)		

	κι∀νο-κ6∀νο=	front-back and high-low vowel contrast (high-low vowel contrast in Kent's list)		
37.	Chop	Chap	Shop	Top
	Τσίρος	**Τσάρος**	**Σύρος**	**Τύρος**
IPA.	∀τσι4οσ	∀τσ64οσ	∀σι4οσ	
	∀τι4οσ			
	∀τσι4οσ-∀τσ64οσ=	front-back and high-low vowel contrast (front-back vowel contrast in Kent's list)		
	∀τσι4οσ-∀σι4οσ=	fricative-affricate contrast		
	∀τσι4οσ-∀τι4οσ=	stop-affricate contrast		
38.	Ship	Sheep	Chip	Tip
	Πίτσα	**Πέτσα**	**Πίσσα**	**Πίτα**
IPA.	∀πιτσ6	∀πΕτσ6	∀πισ6	
	∀πιτ6			
	∀πιτσ6-∀πΕτσ6=	high-mid vowel contrast (vowel duration contrast in Kent's list)		
	∀πιτσ6-∀πισ6=	fricative-affricate contrast		
	∀πιτσ6-∀πιτ6=	stop-affricate contrast		
39.	Feet	Fit	Heat	Fat
	Χωθεί	**Χυθεί**	**Σωθεί**	**Χαθεί**
IPA.	ξο∀Τι	ξι∀Τι	σο∀Τι	ξ6∀Τι
	ξο∀Τι-ξι∀Τι=	front-back and high-mid vowel contrast (vowel duration contrast in Kent's list)		
	ξο∀Τι-σο∀Τι=	other fricative contrast		
	ξο∀Τι-ξ6∀Τι=	mid-low vowel contrast (high-low vowel contrast in Kent's list)		
40.	Coat	Goat	Code	Tote
	Τσάμπα	**Τζάμπα**	**Τσάπα**	**Τάμπα**
IPA.	∀τσ6μβ6	∀τζ6μβ6	∀τσ6π6	
	∀τ6μβ6			
	∀τσ6μβ6-∀τζ6μβ6=	voicing contrast initial consonant		
	∀τσ6μβ6-∀τσ6π6=	voicing contrast medial consonant (voicing contrast final consonant in Kent's list)		
	∀τσ6μβ6-∀τ6μβ6=	stop-affricate contrast (consonant place contrast in Kent's list)		
41.	Dug	Tug	Duck	Bug
	Πήζω	**Μπήζω**	**Πίσω**	**Τήζω**
IPA.	∀πιζο	∀βιζο	∀πισο	∀τιζο
	∀πιζο-∀βιζο=	voicing contrast initial consonant		
	∀πιζο-∀πισο=	voicing contrast medial consonant (voicing contrast final consonant in Kent's list)		
	∀πιζο-∀τιζο=	consonant place contrast		
42.	Cash	Gash	Catch	Cat
	Μπάσα	**Πάσα**	**Μπάτσα**	**Μπάζα**
IPA.	∀β6σ6	∀π6σ6	∀β6τσ6	
	∀β6ζ6			
	∀β6σ6-∀π6σ6=	voicing contrast initial consonant		
	∀β6σ6-∀β6τσ6=	fricative-affricate contrast		
	∀β6σ6-∀β6ζ6=	voicing contrast medial consonant (stop-fricative contrast in Kent's list)		
43.	Fill	Hill	Pill	Full
	Φόρα	**Χώρα**	**Τώρα**	**Φάρα**
IPA.	∀φο46	∀ξο46	∀το46	∀φ646
	∀φο46-∀ξο46=	other fricative contrast		
	∀φο46-∀το46=	stop-fricative contrast		

	∀φo46-∀φ646=	mid-low vowel contrast (front-back vowel contrast in Kent's list)		
44.	Hat	Fat	Pat	That
	Θύμα	**Χύμα**	**Τίμα**	**Σήμα**
IPA.	∀Τιμ6	∀ξιμ6	∀τιμ6	∀σιμ6
	∀Τιμ6-∀ξιμ6=	Other fricative contrast		
	∀Τιμ6-∀τιμ6=	stop-fricative contrast		
	∀Τιμ6-∀σιμ6=	other fricative contrast (alveolar-dental place contrast)		
45.	Hold	Old	Fold	Cold
	Γάμα	**Άμα**	**Βάμμα**	**Γκάμα**
IPA.	∀Γ6μ6	∀6μ6	∀ϖ6μ6	∀γ6μ6
	∀Γ6μ6-∀6μ6=	initial velar-null contrast (initial glottal-null contrast in Kent's list)		
	∀Γ6μ6-∀ϖ6μ6=	other fricative contrast		
	∀Γ6μ6-∀γ6μ6=	stop-fricative contrast		
46.	Heat	Eat	Feet	Hate
	Χάσε	**Άσε**	**Βράσε**	**Χύσε**
IPA.	∀ξ6σE	∀6σE	∀ϖ46σE	
	∀ξισE			
	∀ξ6σE-∀6σE=	initial velar-null contrast (initial glottal-null contrast in Kent's list)		
	∀ξ6σE-∀ϖ46σE=	initial cluster-fricative contrast (other fricative contrast in Kent's list)		
	∀ξ6σE-∀ξισE=	high-low and front-back vowel contrast (high-low vowel contrast in Kent's list)		
47.	Bill	Mill	Dill	Gill
	Λίμπα	**Λίμα**	**Λίντα**	**Λίγκα**
IPA.	∀λιβ6	∀λιμ6	∀λιδ6	∀λιγ6
	∀λιβ6-∀λιμ6=	stop-nasal contrast		
	∀λιβ6-∀λιδ6=	consonant-place contrast		
	∀λιβ6-∀λιγ6=	consonant-place contrast		
48.	Ache	Aches	Ape	Ate
	Εδώ	**Αιδώς**	**Εγώ**	**Ενώ**
IPA.	E∀Δo	E∀Δoσ	E∀Γo	E∀νo
	E∀Δo-E∀Δoσ=	final consonant-null contrast (final cluster-final singleton in Kent's list)		
	E∀Δo-E∀Γo=	consonant place contrast		
	E∀Δo-E∀νo=	fricative-nasal contrast (consonant place contrast in Kent's list)		
49.	Lip	Leap	Lit	Rip
	Λύπη	**Λέπι**	**Λύκοι**	**Ρίποι**
IPA.	∀λιπι	∀λEπι	∀λικι	∀4ιπι
	∀λιπι-∀λEπι=	high-mid vowel contrast (vowel duration contrast in Kent's list)		
	∀λιπι-∀λικι=	consonant place contrast		
	∀λιπι-∀4ιπι=	/r/-/l/ glide-lateral approximant contrast (/r/-/l/ liquid contrast in Kent's list)		
50.	Reap	Rip	Leap	Weep
	Ρένα	**Ρίνα**	**Λένα**	**Ένα**
IPA.	∀4Eν6	∀4ιν6	∀λEν6	∀Eν6
	∀4Eν6-∀4ιν6=	high-mid vowel contrast (vowel duration contrast in Kent's list)		
	∀4Eν6-∀λEν6=	/r/-/l/ glide-lateral approximant contrast (/r/-/l/ liquid contrast in Kent's list)		
	∀4Eν6-∀Eν6=	initial consonant-null contrast (/r/-/w/ contrast in Kent's list)		
51.	Rise	Wise	Lies	Eyes

	Ράμμα	**Νάμα**	**Λάμα**	**Άμα**
IPA.	∀46μ6	∀nθμ6	∀λ6μ6	∀6μ6
	∀46μ6-∀nθμ6=	glide-nasal contrast (/r/-/w/ contrast in Kent's list)		
	∀46μ6-∀λ6μ6=	/r/-/l/ glide-lateral approximant contrast (/r/-/l/ liquid contrast in Kent's list)		
	∀46μ6-∀6μ6=	initial consonant-null contrast		
52.	Row	Woe	Low	Owe
	Ρύπος	**Ρέπος**	**Λίπος**	**Ίππος**
IPA.	∀4ιπoσ	∀4Επoσ	∀λιπoσ	
	∀ιπoσ			
	∀4ιπoσ-∀4Επoσ=	high-mid vowel contrast (/r/-/w/ contrast in Kent's list)		
	∀4ιπoσ-∀λιπoσ=	/r/-/l/ glide-lateral approximant contrast (/r/-/l/ liquid contrast in Kent's list)		
	∀4ιπoσ-∀ιπoσ=	initial consonant-null contrast		
53.	Wax	Wack	Lax	Racks
	Λείψει	**Λύσει**	**Λείπει**	**Ρίψη**
IPA.	∀λιπσι	∀λισι	∀λιπι	∀4ιπσι
	∀λιπσι-∀λισι=	medial double consonant-medial single consonant contrast (final cluster-final singleton contrast in Kent's list)		
	∀λιπσι-∀λιπι=	medial double consonant-medial single consonant contrast (/w/-/l/ contrast in Kent's list)		
	∀λιπσι-∀4ιπσι=	/r/-/l/ glide-lateral approximant contrast (/r/-/w/ contrast in Kent's list)		
54.	Dock	Docks	Mock	Knock
	Μπήξει	**Μπήξεις**	**Μύξη**	**Νύξη**
IPA.	∀βικσι	∀βικσισ	∀μικσι	
	∀νικσι			
	∀βικσι-∀βικσισ=	final consonant-null contrast (final cluster-final singleton contrast in Kent's list)		
	∀βικσι-∀μικσι=	stop-nasal contrast		
	∀βικσι-∀νικσι=	stop-nasal contrast		
55.	Cheer	Sheer	Sear	Tear
	Πάθος	**Πίθος**	**Πάφος**	**Πάχος**
IPA.	∀π6Τoσ	∀πιΤoσ	∀π6φoσ	
	∀π6ξoσ			
	∀π6Τoσ-∀πιΤoσ=	high-low and front-back vowel contrast (fricative-affricate contrast in Kent's list)		
	∀π6Τoσ-∀π6φoσ=	other fricative contrast (fricative-affricate contrast in Kent's list)		
	∀π6Τoσ-∀π6ξoσ=	other fricative contrast (stop-affricate contrast in Kent's list)		
56.	Hash	Hatch	Ash	Dash
	Κάνω	**Κάζο**	**Άνω**	**Χάνω**
IPA.	∀κ6νo	∀κ6ζo	∀6νo	∀ξ6νo
	∀κ6νo-∀κ6ζo=	fricative-nasal contrast (fricative-affricate contrast in Kent's list)		
	∀κ6νo-∀6νo=	initial velar-null contrast (initial glottal-null contrast in Kent's list)		
	∀κ6νo-∀ξ6νo=	stop-fricative contrast		
57.	Tile	Dial	Pile	Mile
	Θήκη	**Δίκη**	**Φύκι**	**Νίκη**
IPA.	∀Τικι	∀Δικι	∀φικι	∀νικι
	∀Τικι-∀Δικι=	voicing contrast-initial consonant		
	∀Τικι-∀φικι=	consonant place contrast		
	∀Τικι-∀νικι=	fricative-nasal contrast (stop-nasal contrast in Kent's list)		
58.	Bunch	Much	Punch	Bun
	Λάπα	**Λάμπα**	**Λάμα**	**Λα**

IPA.	∀λ6π6	∀λ6β6	∀λ6μ6	λ6
	∀λ6π6-∀λ6β6=	voicing contrast-medial consonant (stop-nasal contrast in Kent's list)		
	∀λ6π6-∀λ6μ6=	stop-nasal contrast (voicing contrast initial consonant in Kent's list)		
	∀λ6π6-λ6=	Disyllable-monosyllable contrast (final consonant-null contrast in Kent's list)		
59.	Ease	Is	Cheese	Peas
	Όση	**Ίση**	**Τόση**	**Πόση**
IPA.	∀οσι	∀ισι	∀τοσι	∀ποσι
	∀οσι-∀ισι=	front-back and high-mid vowel contrast (vowel duration contrast in Kent's list)		
	∀οσι-∀τοσι=	initial consonant-null contrast		
	∀οσι-∀ποσι=	initial consonant-null contrast		
60.	Seed	See	Seeds	Feed
	Δές	**Δε**	**Θές**	**Γές**
IPA.	ΔΕσ	ΔΕ	ΤΕσ	ΓΕσ
	ΔΕσ-ΔΕ=	final consonant-null contrast		
	ΔΕσ-ΤΕσ=	voicing contrast initial consonant (final cluster-final singleton contrast in Kent's list)		
	ΔΕσ-ΓΕσ=	other fricative contrast		
61.	Sink	Sing	Pink	Ink
	Ρίψη	**Ρύση**	**Λήψη**	**Ύψη**
IPA.	∀4ιπσι	∀4ισι	∀λιπσι	∀ιπσι
	∀4ιπσι-∀4ισι=	medial double consonant-medial single consonant contrast (final cluster-final singleton contrast in Kent's list)		
	∀4ιπσι-∀λιπσι=	/r/-/l/ glide-lateral approximant contrast (stop-fricative contrast in Kent's list)		
	∀4ιπσι-∀ιπσι=	initial consonant-null contrast		
62.	Harm	Arm	Charm	Farm
	Χήρος	**Ίρος**	**Γύρος**	**Σύρος**
IPA.	∀ξι4οσ	∀ι4οσ	∀Γι4οσ	
	∀σι4οσ			
	∀ξι4οσ-∀ι4οσ=	Initial velar-null contrast (initial glottal-null contrast in Kent's list)		
	∀ξι4οσ-∀Γι4οσ=	voicing contrast initial consonants (fricative-affricate contrast in Kent's list)		
	∀ξι4οσ-∀σι4οσ=	other fricative contrast (alveolar-velar place contrast)		
63.	Cake	Cakes	Take	Ache
	Κόπια	**Κόπιας**	**Τόπια**	**Όποια**
IPA.	∀κοπ 6	∀κοπ 6σ	∀τοπ 6	
	∀οπ 6			
	∀κοπ 6-∀κοπ 6σ=	final consonant-null contrast (final cluster-final singleton contrast in Kent's list)		
	∀κοπ 6-∀τοπ 6=	consonant place contrast		
	∀κοπ 6-∀οπ 6=	initial consonant-null contrast		
64.	Meat	Me	Meats	Neat
	Ποινή	**Πη**	**Ποινής**	**Κοινή**
IPA.	πι∀νι	πι	πι∀νισ	κι∀νι
	πι∀νι-πι=	final syllable-null contrast or disyllable-monosyllable contrast (final consonant-null contrast in Kent's list)		
	πι∀νι-πι∀νισ=	final consonant-null contrast (final cluster-final singleton in Kent's list)		
	πι∀νι-κι∀νι=	consonant place contrast		
65.	Had	Add	Pad	Hid
	Γέλα	**Έλα**	**Γκέλα**	**Γάλα**

IPA.	∀ΓΕλ6	∀Ελ6	∀γΕλ6	∀Γ6λ6
	∀ΓΕλ6-∀Ελ6=	initial velar-null contrast (initial glottal-null contrast in Kent's list)		
	∀ΓΕλ6-∀γΕλ6=	stop-fricative contrast		
	∀ΓΕλ6-∀Γ6λ6=	front-back vowel and mid-low vowel contrast (high-low vowel contrast in Kent's list)		
66.	Hail	Ail	Sail	Tail
	Χήρα	**Ήρα**	**Θήρα**	**Γύρα**
IPA.	∀ξι46	∀ι46	∀Τι46	∀Γι46
	∀ξι46-∀ι46=	initial velar-null contrast (initial glottal-null contrast in Kent's list)		
	∀ξι46-∀Τι46=	other fricative contrast		
	∀ξι46-∀Γι46=	voicing contrast initial consonant (stop-fricative contrast in Kent's list)		
67.	Hall	All	Tall	Ball
	Χώρος	**Όρος**	**Πόρος**	**Ντόρος**
IPA.	∀ξο4οσ	∀ο4οσ	∀πο4οσ	
	∀δο4οσ			
	∀ξο4οσ-∀ο4οσ=	initial velar-null contrast (initial glottal-null contrast in Kent's list)		
	∀ξο4οσ-∀πο4οσ=	stop-fricative contrast		
	∀ξο4οσ-∀δο4οσ=	stop-fricative contrast		
68.	Fork	Four	Forks	Cork
	Στις	**Στη**	**Στήσε**	**Τις**
IPA.	στισ	στι	∀στισΕ	τισ
	στισ-στι=	final consonant-null contrast (final cluster-final singleton contrast in Kent's list)		
	στισ-∀στισΕ=	final vowel-null contrast (final cluster-final singleton contrast in Kent's list)		
	στισ-τισ=	initial cluster-initial singleton contrast (stop-fricative contrast in Kent's list)		
69.	Rake	Ray	Rakes	Lake
	Ρήξη	**Ρη**	**Ρείκι**	**Λήξη**
IPA.	∀4ικσι	4ι	∀4ικι	∀λικσι
	∀4ικσι-4ι=	disyllable-monosyllable contrast (final consonant-null contrast in Kent's list)		
	∀4ικσι-∀4ικι=	medial double consonant-medial single consonant contrast (final cluster-final singleton contrast in Kent's list)		
	∀4ικσι-∀λικσι=	/r/-/l/ glide-lateral approximant contrast (/r/-/l/ liquid contrast in Kent's list)		
70.	Leak	Lee	Leaks	Luke
	Τόσο	**Το**	**Τόσος**	**Τάσο**
IPA.	∀τοσο	το	∀τοσοσ	
	∀τ6σο			
	∀τοσο-το=	final syllable-null contrast or disyllable-monosyllable contrast (final consonant-null contrast in Kent's list)		
	∀τοσο-∀τοσοσ=	final consonant-null contrast (final cluster-final singleton contrast in Kent's list)		
	∀τοσο-∀τ6σο=	high-low vowel contrast (front-back vowel contrast in Kent's list)		

Appendix F

Comparison between the Greek Intelligibility List and Kent's List

SAME PARAMETERS

Greek List	Number of Occur.	Kent's List	Number of Occur.
1. Consonant place contrast	8	Consonant place contrast	9
2. Final consonant-null contrast	10	Final consonant-null contrast	9
3. Final cluster-final singleton contrast	1	Final cluster-final singleton contrast	12
4. Fricative-affricate contrast	5	Fricative-affricate contrast	9
5. Front-back vowel contrast	5	Front-back vowel contrast	11
6. High-low vowel contrast	2	High-low vowel contrast	12
7. Initial consonant-null contrast	14	Initial consonant-null contrast	14
8. Initial cluster-null contrast	1		
9. Initial cluster-initial singleton contrast	11	Initial cluster-initial singleton contrast	12
10. Other fricative contrast	16	Other fricative contrast	15
11. Stop-affricate contrast	4	Stop-affricate contrast	6
12. Stop-fricative contrast	16	Stop-fricative contrast	20
13. Stop-nasal contrast	6	Stop-nasal contrast	10
14. Voicing contrast initial consonant	11	Voicing contrast initial consonant	9
15. Voicing contrast final consonant	1	Voicing contrast final consonant	11

DIFFERENT PARAMETERS

16. Alveolar-dental place contrast	2		
17. Alveolar-labiodental place contrast	1		
18. Alveolar-velar place contrast	3	Alveolar-palatal place contrast	8
19. Bilabial-velar place contrast	1		
20. Disylable-monosyllable contrast	5		
21. Fricative-nasal contrast	4		
22. Front-back and high-low vowel contrast	10		
23. Front-back and mid-low vowel contrast	1		
24. Glide-nasal in a cluster contrast	1		
25. Glide-nasal contrast	2		
26. Glide-fricative contrast	4		
27. High-mid vowel contrast	12	Vowel duration contrast	11
28. Initial cluster-fricative contrast	1		
29. Initial cluster (3)-initial cluster (2) contr.	2		
30. Initial velar-null contrast	11	Initial glottal-null contrast	11
31. Labiodental-dental place contrast	1		
32. Medial double consonant-medial single consonant contrast	4		
33. Mid-low vowel contrast	4		
34. /r/-/l/ (glide-lateral approximant contrast)	11	/r/-/l/ contrast	10
35. Voiced-voiceless fricative contrast	2		
36. Voicing contrast medial consonant	12		
37. Lateral-nasal contrast	1	/r/-/w/ contrast	8
38. Final vowel-null contrast	1		
39. Front-back and high mid vowel contrast	3		
Total number of parameters:	39	Total number of parameters:	19

Appendix G

List of words for intelligibility estimation

1.	ˈpizo	ˈpɛzo	ˈpiso	ˈbizo
2.	ˈxomɐ	ˈsomɐ	ˈkomɐ	ˈɣomɐ
3.	ˈpliθos	ˈpiθos	ˈliθos	ˈiθos
4.	ˈnotɐ	ˈrotɐ	ˈnondɐ	ˈnɐtɐ
5.	poˈso	poˈθo	poˈto	poˈsu
6.	ˈxitɐ	ˈsitɐ	ˈθitɐ	ˈitɐ
7.	ˈkrimɐ	ˈkimɐ	ˈrimɐ	ˈkrimɐs
8.	tu	to	ti	tɐ
9.	ˈlisɐ	ˈlusɐ	ˈlizɐ	ˈrisɐ
10.	ˈtsimɐ	ˈsimɐ	ˈtimɐ	ˈimɐ
11.	ˈmɐsɐ	ˈmɐzɐ	ˈmɐtɐ	ˈbɐsɐ
12.	ˈrisi	ˈrizi	ˈlisi	ˈðisi
13.	ˈmɐgɐ	ˈmɐnɐ	ˈmɐkɐ	mɐ
14.	θɐ ˈsoso	θɐ ˈzoso	θɐ ˈsiso	θɐ ˈsozo
15.	ˈxɐli	ˈɐli	ˈsɐli	ˈzɐli
16.	ˈiði	ˈɣiði	ˈiθi	ˈfiði
17.	ˈlitsɐ	ˈlisɐ	ˈnitsɐ	ˈlitɐ
18.	ˈkɐzo	ˈkɐdo	ˈkɐno	ˈkɐro
19.	ˈðɛno	ˈzɛno	ˈdɛno	ˈvɛno
20.	bis	bɐs	biz	bi
21.	ˈpirɐ	ˈpɛrɐ	ˈpɐrɐ	ˈpurɐ
22.	ˈiðɛs	ˈʁiðɛs	ˈviðɛs	ˈiθɛs
23.	ˈɛnɐ	ˈxɛnɐ	ˈpɛnɐ	ˈɐnɐ

24.	ˈvirɐ	ˈvɛrɐ	ˈvɐrɐ	ˈfirɐ
25.	ˈrimɐ	ˈlimɐ	ˈximɐ	ˈrɛmɐ
26.	ˈzonɔ	ˈɣonɔ	ˈdonɔ	ˈsonɔ
27.	ˈθrɛmɐ	ˈθɛmɐ	ˈrɛmɐ	ˈɛmɐ
28.	ˈkitɐ	ˈpitɐ	ˈkutɐ	ˈkotɐ
29.	ˈθirɐ	ˈfirɐ	ˈxirɐ	ˈtirɐ
30.	ˈprɐsɔ	ˈpɐsɔ	ˈrɐsɔ	ˈprɔsɔ
31.	ˈtrimɐ	ˈtmimɐ	ˈtimɐ	ˈrimɐ
32.	ˈstrivi	ˈtrivi	ˈstivi	ˈstrivis
33.	ˈkiti	ˈkɛti	ˈkɛti	ˈxiti
34.	ˈsolɐ	ˈfolɐ	ˈolɐ	ˈtolɐ
35.	ˈrɐbɐ	ˈzɐbɐ	ˈlɐbɐ	ˈrɐbɐs
36.	kiˈno	kuˈno	kɛˈno	kɐˈno
37.	ˈtsiros	ˈtsɐros	ˈsiros	ˈtiros
38.	ˈpitsɐ	ˈpɛtsɐ	ˈpisɐ	ˈpitɐ
39.	xoˈθi	xiˈθi	soˈθi	xɐˈθi
40.	ˈtsɐmbɐ	ˈtzɐmbɐ	ˈtsɐpɐ	ˈtɐmbɐ
41.	ˈpizo	ˈbizo	ˈpiso	ˈtizo
42.	ˈbɐsɐ	ˈpɐsɐ	ˈbɐtsɐ	ˈbɐzɐ
43.	ˈforɐ	ˈxorɐ	ˈtorɐ	ˈfɐrɐ
44.	ˈθimɐ	ˈximɐ	ˈtimɐ	ˈsimɐ
45.	ˈɣɐmɐ	ˈɐmɐ	ˈvɐmɐ	ˈgɐmɐ
46.	ˈxɐsɛ	ˈɐsɛ	ˈvrɐsɛ	ˈxisɛ
47.	ˈlibɐ	ˈlimɐ	ˈlidɐ	ˈligɐ

48.	ɛˈðo	ɛˈðos	ɛˈɣo	ɛˈno
49.	ˈlipi	ˈlɛpi	ˈliki	ˈɾipi
50.	ˈɾɛnɐ	ˈɾinɐ	ˈlɛnɐ	ˈɛnɐ
51.	ˈɾɐmɐ	ˈnɐmɐ	ˈlɐmɐ	ˈɐmɐ
52.	ˈɾipos	ˈɾɛpos	ˈlipos	ˈipos
53.	ˈlipsi	ˈlisi	ˈlipi	ˈɾipsi
54.	ˈbiksi	ˈbiksis	ˈmiksi	ˈniksi
55.	ˈpɐθos	ˈpiθos	ˈpɐfos	ˈpɐxos
56.	ˈkɐno	ˈkɐzo	ˈɐno	ˈxɐno
57.	ˈθiki	ˈðiki	ˈfiki	ˈniki
58.	ˈlɐpɐ	ˈlɐbɐ	ˈlɐmɐ	lɐ
59.	ˈosi	ˈisi	ˈtosi	ˈposi
60.	ðɛs	ðɛ	θɛs	ɣɛs
61.	ˈɾipsi	ˈɾisi	ˈlipsi	ˈipsi
62.	ˈxiɾos	ˈiɾos	ˈɣiɾos	ˈsiɾos
63.	ˈkopʲɐ	ˈkopʲɐs	ˈtopʲɐ	ˈopʲɐ
64.	piˈni	pi	piˈnis	kiˈni
65.	ˈɣɛlɐ	ˈɛlɐ	ˈgɛlɐ	ˈɣɐlɐ
66.	ˈxiɾɐ	ˈiɾɐ	ˈθiɾɐ	ˈɣiɾɐ
67.	ˈxoɾos	ˈoɾos	ˈpoɾos	ˈdoɾos
68.	stis	sti	ˈstisɛ	tis
69.	ˈɾiksi	ɾi	ˈɾiki	ˈliksi
70.	ˈtoso	to	ˈtosos	ˈtɐso

Appendix H

tɐ ɛlini'kɐ ni'sçɐ o'ktovrios 'xilʝɐ ɛɲɐ'kosçɐ ɛnɛ'nindɐ ɛ'nɛɐ

i ɛ'lɐðɐ os 'xorɐ 'ɛɣinɛ ɣno'sti turisti'kɐ| prin ɐ'po tri'endɐ 'xronɲɐ| ɛksɛ'ties ton ni'sçon tis‖ ɐ'ftɐ 'inɛ pɛ'ripu 'xilʝɐ| ɐn kɛ 'liɣɐ ketі'kundɛ‖ i zo'i ɛ'ki pro'sfɛrɛtɛ ʝɐ 'olus| kɛ ʝɐ ɐ'ftus pu 'θɛlun irɛ'miɐ ɐ'kuɣondɐs 'mono tɐ dzi'dzikçɐ| ɐ'lɐ kɛ ʝɐ 'opʝus 'θɛlun nɐ 'vlɛpun 'kozmo‖

tɐ ni'sçɐ tu ɛ'ɣɛu 'inɛ ksɛ'rɐ mɛ liɣo'stɐ 'ðɛndrɐ| ɛ'no tɐ plɐi'nɐ tu io'niu 'ɛxun po'li xlo'riðɐ‖ 'olɐ tɐ ni'sçɐ 'ɛxun 'rɛmɐtɐ| 'omvriɐ nɛ'rɐ kɛ vu'nɐ pu 'ftɐnun sɛ mɛ'ɣɐlo 'ipsos‖ sɛ 'olɐ 'ɛzisɐn kɛ zun 'oxi 'mono 'spɐniɐ pti'nɐ ɐ'lɐ kɛ ki'nɐ 'zoɐ| 'opos i 'kotɐ| i 'ɣɐtɐ kɛ i kɐ'tsikɐ‖ sɛ 'ɛnɐ ni'si 'vriken 'ɛnɐ 'iðos 'zou monɐði'ko 'pɐno sti ɣi| to kri'kri‖

tɐ mɛ'ɣɐlɐ ni'sçɐ 'inɛ 'mono 'pɛndɛ kɛ 'ɛxun 'ɐfθonɛs ɐ'ktɛs| pu 'kɐnun ton 'ksɛno nɐ rɛ'mvɐzi sɐ'fto to 'θɐvmɐ tis 'fisis‖ ɛ'ðo| ðɛn θɐ vris puθɛ'nɐ 'utɛ tis ɐ'prositɛs orosi'rɛs 'utɛ tis 'polis mɛ tin 'molinsi| tɐ 'gɛto kɛ tɐ 'ɐtsɐlɐ 'ɐxromɐ 'ktiri'ɐ tus‖ ɐn 'θɛlis 'vɛvɛɐ| bo'ris nɐ vris sxɛ'ðon pɐ'ndu sɛ 'olɐ tɐ ni'sçɐ 'kɐti 'ɐɣnostɛs ston po'li 'kozmo ɐmu'ðçɛs| iði'kɐ stis 'piso plɛ'vrɛs tus‖ 'totɛ θɐ ɐpo'lɐfsis ti 'ðisi tu 'iʌʝu| ɛ'ki pu 'zmiɣi o urɐ'nos mɛ ti 'θɐlɐsɐ‖ ɐpo'pu 'ɐrɐɣɐ ɛ'mbnɛfstikɛ o so'fos θɛ'os ʝɐ nɐ 'plɐsi ɐ'fti tin pɛmptu'siɐ tis omo'rfçɛs‖

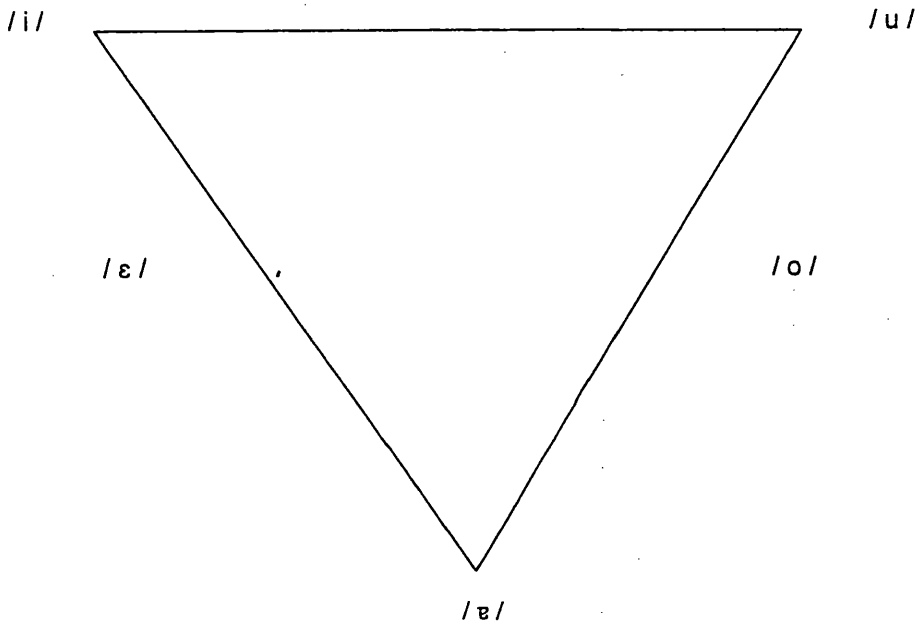
sɛ 'olɐ tɐ mi'krɐ ni'sçɐ o 'kozmos 'inɛ si'niθos ndropɐ'los ɐ'lɐ ɛnðiɐ'fɛrɛtɛ nɐ ɛfxɐri'stisi ton ɐpi'skɛpti‖ 'kɐpʝi 'dinondɛ mɛ tɐ kɐ'lɐ tus 'ruxɐ 'otɐn 'ɛrxondɛ 'ksɛni‖ mɛri'ki fo'nɐzun| kɐ'los ton 'ksɛno‖ 'ɛlɐ 'kɐtsɛ nɐ 'pçis 'kɐti‖ ti nɐ sɛ fi'lɛpsumɛ‖

i 'kɐtiki ton ni'sçon 'inɛ ɐ'pli‖ pi'stɛvun stin 'ɐspili mo'rfi tis pɐnɐ'ɣiɐs‖ ɐ'komɐ kɛ 'torɐ ɐ'kuɣɛtɛ 'oti sto 'tɐðɛ ni'si 'ɛɣinɛ 'θɐvmɐ‖ pɐ'lçɐ 'itɐn 'mono psɐ'rɐðɛs‖ 'simɛrɐ mɛ tin 'ɐfksisi tu turi'smu o'θundɛ kɛ sɛ 'ɐlɛs ɐsxo'liɛs‖ si'xnɐ 'ɣinondɛ nɐ'fti'ki kɛ ipirɛ'tun sɛ 'sinxronɐ ɛbori'kɐ 'plɐ| pu 'skizun tus okɛɐ'nus‖ 'tɛlos| 'ɐli 'pçɐnunɛ sfu'gɐrʝɐ| kɛ 'ɐli ɛ'ktrɛfun 'provɐtɐ kɛ 'ɣiðʝɐ‖

pɐ'lçɐ| 'osɐ ni'sçɐ ðɛn kɐ'tixɐn i 'frɐgi kɛ i ɐnɛ'ti| 'isɐn ormi'tiriɐ pirɐ'ton i o'pii 'oryonɐn ti mɛ'soɣio 'θɐlɐsɐ‖ ɐ'fti xrisimo'piusɐn tɐ ni'sçɐ ɐ'ftɐ sɐn mo'xlus tis kiriɐ'rxiɐs tus‖ kɐ'tɛplɛɐn sɛ ɐ'prositɛs ɐ'ktɛs| 'ɛspɛrnɐn simfo'rɛs| 'ɛvlɛptɐn tis θɐ'lɐsiɛs sigino'niɛs| 'vulʝɐzɐn 'plɐ| sko'rpusɐn ton 'tromo| ki'nundɐn ipo'xθoniɐ kɛ fθonɛ'rɐ‖ i 'dopʝi 'ixɐn 'sθɛnos| ɐ'lɐ i pirɐ'tɛs 'ɛlɛnxɐn tin kɐ'tɐstɐsi kɛ 'tsɐkizɐn 'kɐθɛ ɐ'distɐsi‖ ɛ'ki 'opu kiriɐ'rxusɐn i 'frɐgi 'sozondɛ 'simɛrɐ tɐ 'kɐstrɐ mɛ tɐ ɛ'mvlimɐ'tɐ tus kɛ 'ixni ɐ'po tɐ 'volʝɐ tus‖

Appendix I

The Greek vowels



Appendix J

Combination of vowels in the reading passage

[i] + Other Vowels

[ii] → Δύση, Φύσης

[iε] → είναι

[iu] → Not found

[io] → είδος, ύψος

[ια] → ήταν, λίγα, μικρά

[u] + Other Vowels

[ui] → Not found

[uε] → ούτε

[uu] → Not found

[uo] → Not found

[ua] → βουνά, ρούχα

[ε] + Other Vowels

[εi] → εκεί

[εε] → πέντε

[ευ] → έχουν, θέλουν

[εο] → γκέτο

[εα] → πέρα

[ο] + Other Vowels

[οι] → ότι, όχι, πολύ, ζωή

[οε] → τότε

[ου] → όλους, ζώου, αυτούς όπου

[οο] → όπως, κόσμο

[οα] → ζώα, κότα, χώρα

[α] + Other Vowels

[αi] → άλλοι, κάτι, πλάσει

[αε]→ άλλες, τάδε

[αυ] → αυτούς, κάνουν, παντού, φτάνουν

[αο] → από, αυτό

[αα] → αυτά, άλλα, θαύμα

Appendix K

Vowels and Consonants in the reading passage

Phonetic Symbol	Initial	Medial	Final
[i]	**η** (4), **είναι** (2), γίνονται, πίσω, **οι** (3), **ήταν** (2)	της (3), την, πτηνά, περίπου	κρι, τη, εκεί κάτι
[ε]	ένα, ενώ, έχουν (2), έγινε πέντε, βέβαια, εκεί, έλα	βέβαια, πουθενά	και (2), άραγε τάδε, τότε
[u]	πουθενά, ούτε (2)	έχουν, πεμπτουσία ακούγοντας	του (2), Αιγαίου, περίπου, από που αυτούς, που παντού
[o]	ότι, όπως, ως, όλους, όλα (3), οποίοι, όταν, όσα, όποιους, όπου, όχι, πολύ, ο (3), κότα, σοφός, τώρα	τον, στον, απολαύσεις	είδος, αυτό σοφός, το στο, από
[α]	κάτι, από (3), γάτα, αν ακτές, αλλά, τάδε απλοί, άλλες	μεγάλα, όταν, ήταν	κότα, να, τα (2), όλα, γάτα, κατσίκα, αυτά
		DIPTHONGS	
[αι]		πλαινά	
[οι]			
		OTHER DIPTHONGS	
[ιι]			κάποιοι
[ιου]		όποιους	ήλιου
[ιο]		νησιών	
[ια]			παλιά, νησιά (2)
		CONSONANTS Plosives	
[p]	περίπου, πολύ, που (2), πάνω	βλέπουν όπως	περίπου
[b]	μπορείς	εμπορικά	-
[t]	του, τη, της, τα (3) τους, των	τότε, όταν κότα, γάτα ούτε	-

[d]	ντύνονται	πέντε, παντού	γίνονται κατοικούνται
[k]	και (4), κάνουν, κότα, κοινά, κάτι, κατσίκα	εκεί	κατσίκα
[g]	γκέτο	συγκοινωνίες	-
		Nasals	
[m]	με (2), μόνο (2)	ηρεμία	-
[n]	να, νησιών, νερά	είναι (2), ενώ	αν, έχουν ήταν, δεν πριν
		Trill	
[r]	ρέματα, ρούχα	νερά, χώρα	σήμερα
		Affricates	
[ts]	-	άτσαλα, κατσίκα κάτσε	-
[tz]	τζιτζίκια	τζιτζίκια	-
		Fricatives	
[f]	φύσης	σοφός	-
[v]	βουνά	πρόβατα	-
[θ]	θέλεις, θέλουν θα, θεός	ωθούνται, πουθενά	- συνήθως
[ð]	δέντρα, δεν (1) δύση	είδος, εδώ ειδικά	Ελλάδα
[s]	σε (3), σαν, συχνά	νησιών, δύση νησιά, νησί,	πόλεις, ως, της, τους, καλώς, αυτούς, όπως, συνήθως
[z]	ζωή, ζουν, ζώα, ζώου	έζησαν	-
[χ]	χίλια, χώρα	έχουν (1), όχι είχαν	-
[γ]	για (2), γίδια, γάτα, γίνονται γη	λίγα, έγινε, ακούγοντας Αιγαίου	άραγε
		Lateral Approximant	
[l]	λίγα	όλα (2), χίλια	μεγάλα

		αλλά, πολύ, όλους, άλλες πόλεις	
		Double Consonants	
[x]	ξένο, ξερά, ξένοι	εξαιτίας	-
[ps]	ψαράδες	ύψος φιλέψουμε	-
		Consonant Blends	
		Plosives	
[pt]	πτηνά	-	επισκέπτη
[bn]	-	εμπνεύστηκε	-
[mpt]	-	πεμπτουσία	-
[kt]	κτίρια	ακτές (1)	-
[ktr]	-	εκτρέφουν	-
[gt]	-	-	-
		Fricatives	
[fθ]	φθονερά	άφθονες	-
[fx]	-	αύξηση	-
[fs]	-	-	θα απολαύσεις
[fst]	-	εμπνεύστηκε	-
[ft]	φτάνουν	αυτοί, αυτούς ναυτικοί, αυτά	-
[fχ]	-	ευχαριστήσει	-
[vm]	-	θαύμα	-
[sθ]	σθένος	-	-
[sk]	σκίζουν	επισκέπτη	-
[sp]	σπάνια	άσπιλη, έσπερναν	-
[st]	στη, στον στις	-	λιγοστά
[sf]	σφουγγάρια	προσφέρεται	-
[sχ]	σχεδόν	ασχολίες	-
[zm]	σμίγει	κόσμο, κόσμος	τουρισμού
[χθ]	-	υποχθόνια	-
[χn]	-	συχνά, ίχνη	-

[χt]	-	-	-
[γn]	γνωστή	άγνωστες	-
		Lateral Approximant	
[lm]	-	-	επαγγέλματα
		Trill	
[rp]	-	σκορπούσαν	-
[rγ]	-	όργωναν	-
[rn]	-	έσπερναν	-
[rm]	ορμητήρια	-	-
[rf]	-	μορφή ομορφιάς	-
[rχ]	-	έρχονται, κυριαρχίας	-
		Nasals	
[mv]	-	ρεμβάζει	-
[mvl]	-	εμβλήματα	-
[mvr]	-	όμβρια	-
[mf]	-	συμφορές	-
[nð]	-	ενδιαφέρεται	-
[nðr]	-	-	-
[ndr]	ντροπαλός	δέντρα	-
[ns]	-	-	μόλυνση
		Consonants with [l] and [r]	
[pl]	πλάσει	κατέπλεαν απλοί	-
[pr]	προσφέρεται, πριν	απρόσιτες	-
[vr]	-	-	-
[tl]	-	-	-
[tr]	τριάντα, τρόμο	-	-
[ðl]	-	-	-
[ðr]	-	-	-

[kl]	-	-	-
[kr]	κρι κρι	μικρά	-
[γl]	-	-	-
[γr]	-	-	-
[fl]	-	-	-
[fr]	Φράγγοι	-	-
[vl]	βλέπουν	έβλαπταν	-
[vr]	βρήκαν, βρεις	πλευρές	-
[tl]	-	-	-
[sl]	-	-	-
[str]	-	κάστρα	-
[zl]	-	-	-
[χl]	-	μοχλούς	-
[χr]	χρόνια	άχρωμα	-
[γl]	-	-	-

Appendix L

THE INTERNATIONAL PHONETIC ALPHABET (revised to 1993)

GREEK – ENGLISH CONNECTIONS[14]

	Bilabial	Labiodental	Dental	Alveolar	Postalveolar	Palatal	Velar
Plosive	p b **p b**			t d **t d**			k g **k g**
Nasal	m **m**			n **n**			ŋ **ŋ**
Tap or Flap				ɾ			
Affricates				ts dz **ts dz**	tʃ dʃ		
Fricative	f v **f v**		θ ð **θ ð**	s z **s z**			x ɣ
Approximant	w					j	
Lateral Approximant				l **l**			

[14] With boldness stand the Greek consonant symbols and with plain stand the corresponding basic English symbols.

Appendix M

2nd MEETING

Recording Protocol
Parkinson's Project

Subject Number / Initials _____

Date _____

A. Preparation: Take

1. Marrantz CP430 Tape recorder _____
2. Marrantz adaptor _____
   One side in DC Input other side electricity
3. Headphones _____
   Phones
4. Microphone _____
   Stereo/L
5. A blank tape TDK SA60 _____
6. A battery for the microphone _____
7. Frenchay Dysarthria Assessment guidelines _____
8. Frenchay Dysarthria Scoring graph _____
9. Tongue depressor _____
10. Stop watch _____

ADMINISTER THE FRENCHAY DYSARTHRIA ASSESSMENT

ADJUSTMENTS

1. Tape recorder from the left to the right
   - *Speaker* → On _____
   - *Speaker* → L + R (ST) _____
   - *Monitor volume* → Middle position _____
   - *Monitor* → Source position _____
   - *Limiter* → Off _____
   - *Rec Volume* → adjusted according to the case _____
     (Left is the minimum)
   - *Mic ATT* → 0 dB _____
   - *Microphone* → Stereo/L _____
   - *Mic mode* → Stereo _____
   - *Pitch* → 0 position _____
   - *Bias Fine* → 0 position _____
   - *N.R. (noise reduction)* → Off _____
   - *Tape* → CrO2 _____
   - *MPX Filter* → Off _____

- *Memory REW* → Off _____

2. Cards _____

TAPE-RECORDING PROTOCOL

1. Insert a blank CrO2 tape _____

2. Push the *Rec* button and the *Play* button together. When needed The *Pause* button also. _____

3. Be careful not to have the *Peak light* → On during recording. _____

4. The first thing I am going to ask you to do is to <u>count</u> to ten for me:
   a. 1-10 do it again _____
   b. 1-10 one more time _____
   c. 1-10 **CONTROL REC VOLUME NOT RED** _____

5. Read the cards with the words in a normal level (as you regularly talk). Do not care if some of the words do not have a meaning. Read whatever you see. _____

AFTER RECORDING

1. Mark cassette with Date _____
   Subject initials (Surname first and then name) _____

3rd MEETING

Recording Protocol
Parkinson's Project

Subject Number / Initials _____

Date _____

A. Preparation: Take

1. Oscilloscope _____
2. Oscilloscope's adaptor "Thandar" _____
3. Oscilloscope's cord connecting it with ELG _____

   Y Input *Waveform*
4. Laryngograph _____
5. Laryngograph's processor battery charger _____
6. A cord connecting battery charger with electricity _____
7. A cord from Laryngograph to DAT recorder _____

   Tape In/Out *Line In (3 black-3 red)*

   (4 yellow- 4 white)

   Tape In/Out *Line Out (1 red-1 black)*

   (2 white-2 red)
8. A cord with 2 electrodes from the laryngograph _____

   Lx
9. A microphone from the laryngograph _____

   Speech
10. A DAT recorder _____
11. A Sony adaptor from the DAT recorder _____

    DC In 6V to electricity
12. A set of headphones _____

    Phones/Line Out
13. A blank tape for the DAT _____

ADJUSTMENTS

1. Oscilloscope from the Left to the Right
   - *V/DIV* → 5 (the amplitude level /high or low signal) _____
   - *Blue AC button* → pushed _____
   - *Blue DC button* → NOT pushed _____
   - *White AC button* → NOT pushed _____
   - *White DC button* → pushed _____
   - *Trigger Level* → above the – (controls the stability of the signal/ to look like better) _____
   - *White BL button* → pushed _____
   - *White ECON button* → NOT pushed _____

- *Blue TRIG SWEEP button* → pushed _____
- *Blue m sec button* → pushed _____
- *TIME/DIV button*→ 5 (controls how many hills are per rectangle) _____

2. ELG from the Left to the Right
   - *INPUT* → Live _____
   - *OUTPUT* → Lx _____
   - *Green adjuster* → Adjusted _____
   - *Blue adjuster* → Adjusted _____
   - *Lx* → Left (same as Sp) _____
   - *Sp* → Left (same as Sp) _____

3. DAT Recorder from the Left to the Right
   - *AVLS* → On _____
   - *REC MODE* → Manual _____

TAPE-RECORDING PROTOCOL

1. Turn on Oscilloscope, ELG, DAT recorder _____

2. The first thing I am going to ask you to do is to count to ten for me:
   a. 1-10 do it again _____
   b. 1-10 one more time _____
   c. 1-10 **CONTROL REC LEVEL UNTIL 12** _____

3. The next thing I'll ask you to do is to repeat as fast as you can "papapa" until I'll tell you to stop:
   a. "papapa" do it again _____
   b. "papapa" one more time _____
   c. "papapa" _____

4. Say "tatata" as fast as you can
   a. "tatata" do it again _____
   b. "tatata" one more time _____
   c. "tatata" _____

5. Say "kakaka" as fast as you can
   a. "kakaka" do it again _____
   b. "kakaka" one more time _____
   d. "kakaka" _____

6. Say "pataka" as fast as you can
   a. "pataka" do it again _____
   b. "pataka" one more time _____
   c. "pataka" _____

7. Now I am going to ask you to do sustained phonation. It is to hold an "a" without a change of pitch:
   a. "aaaaaa" do it again _____
   b. "aaaaaa" one more time _____
   c. "aaaaaa" _____

8. Now I am going to ask you to do sustained phonation. It is to hold an "u" without a change of pitch:
   a. "uuuuuu" do it again _____
   b. "uuuuuu" one more time _____

c. "uuuuuu" _____

9. Now I am going to ask you to do sustained phonation. It is to hold an "i" without a change of pitch:
   a. "iiiiiii" do it again _____
   b. "iiiiiii" one more time _____
   c. "iiiiiii" _____

10. Now I am going to ask you to do sustained phonation. It is to hold an "ε" without a change of pitch:
   a. "εεεεε" do it again _____
   b. "εεεεε" one more time _____
   c. "εεεεε" _____

11. Now I am going to ask you to do sustained phonation. It is to hold an "o" without a change of pitch:
   a. "oooooo" do it again _____
   b. "oooooo" one more time _____
   c. "oooooo" _____

12. Now we are going to read a passage. It is a story that has most of the sounds of the Greek language _____

13. Now we are going to talk a little bit about the earthquakes. What was your experience about an earthquake? _____

AFTER RECORDING

1. Mark cassette with Date _____
   Subject initials/number _____

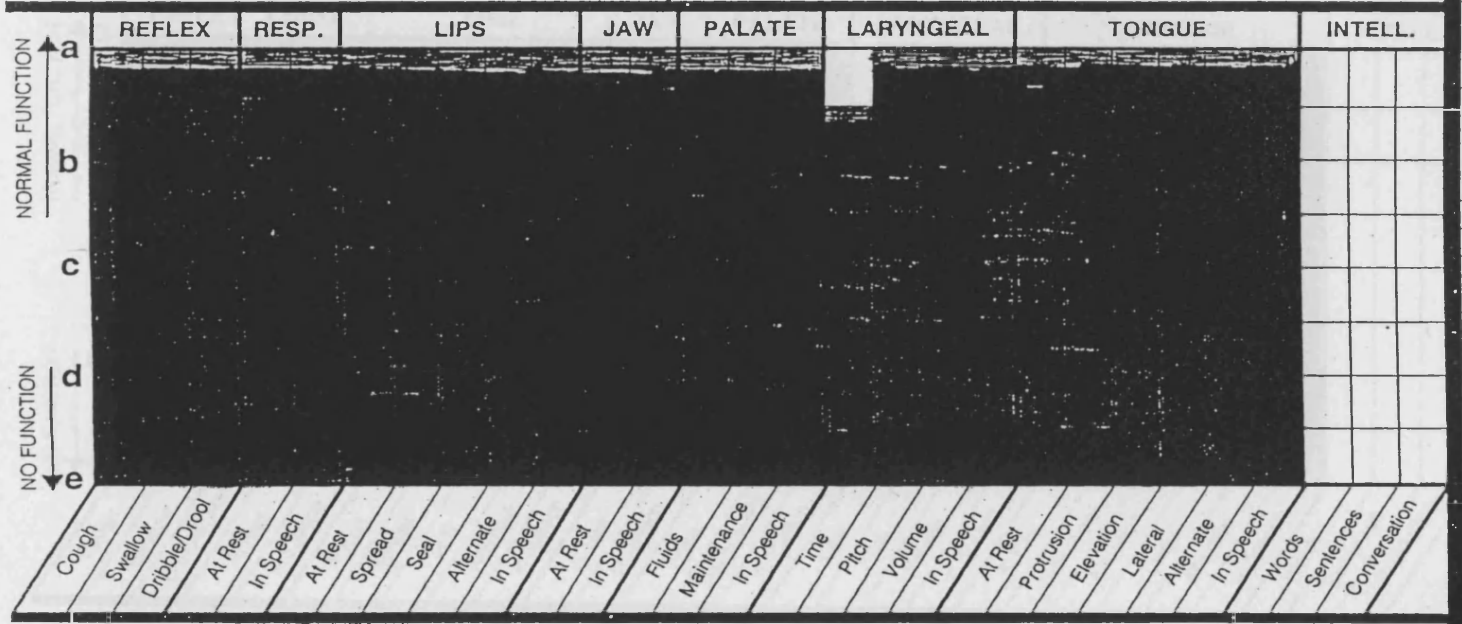
Appendix N

Copies of the completed Frenchay Dysarthria Assessment forms of all subjects

FRENCHAY DYSARTHRIA ASSESSMENT
SCORING FORM
SPEECH THERAPY

FAMILY NAME
FIRST NAME/S
ADDRESS
DATE OF BIRTH

NAME OF THERAPIST KK
CLIENT NUMBER BE #1 Control
DATE OF ASSESSMENT 23/1/2000



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder ×

HEARING
SIGHT
TEETH
LANGUAGE
MOOD
POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R
UPPER LIP L
TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FRENCHAY DYSARTHRIA ASSESSMENT

SCORING FORM

SPEECH THERAPY

NELSON

FAMILY NAME

FIRST NAME/S

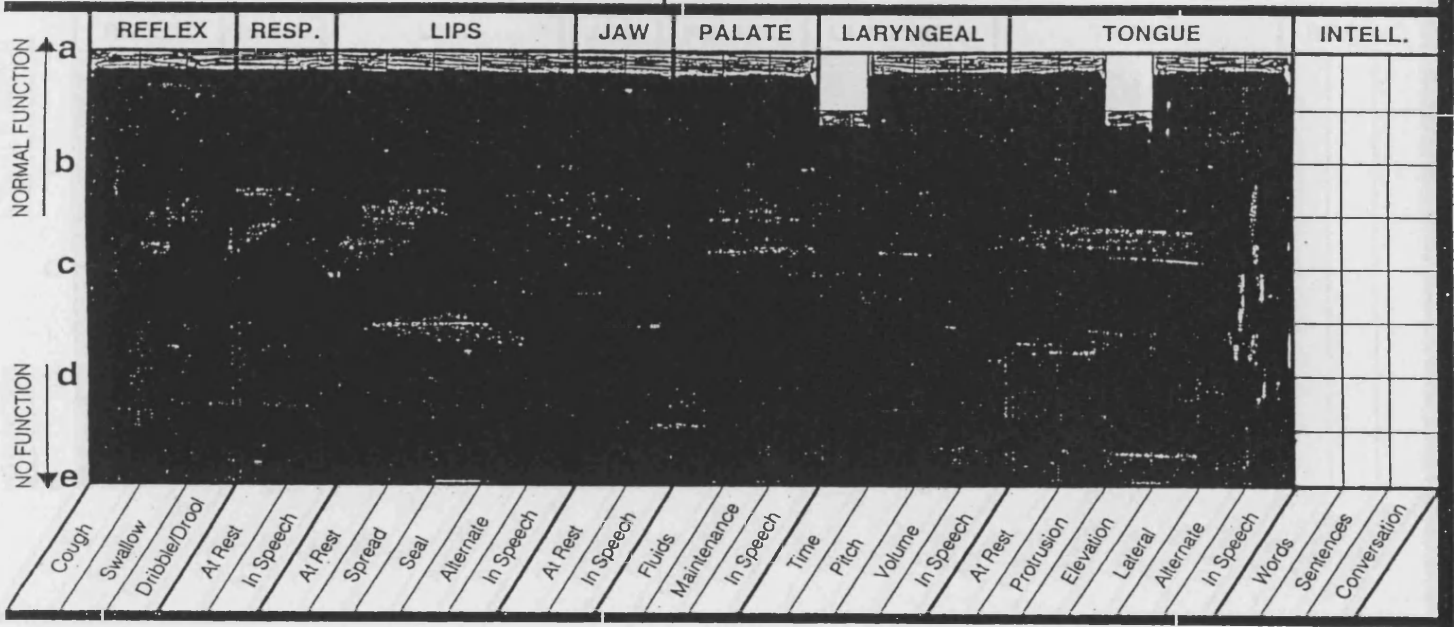
ADDRESS

DATE OF BIRTH

NAME OF THERAPIST KK

CLIENT NUMBER AN #2 Control

DATE OF ASSESSMENT 16/5/2000



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS

Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FRENCHAY DYSARTHRIA ASSESSM[illegible]T

NELSON

FAMILY NAME

FIRST NAME/S

ADDRESS

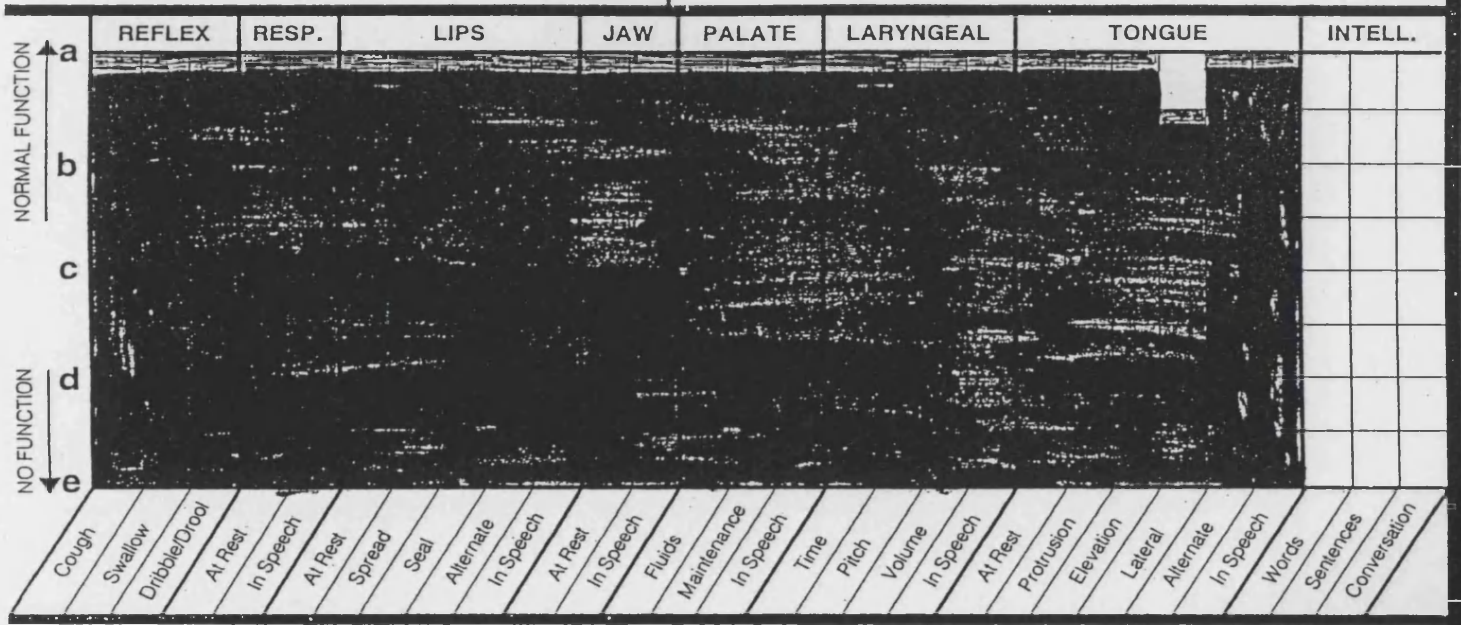
DATE OF BIRTH

SCORING FORM
SPEECH THERAPY

NAME OF THERAPIST KK

CLIENT NUMBER BA #3 Control

DATE OF ASSESSMENT 20/5/2000



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

FRENCHAY DYSARTHRIA ASSESSMENT

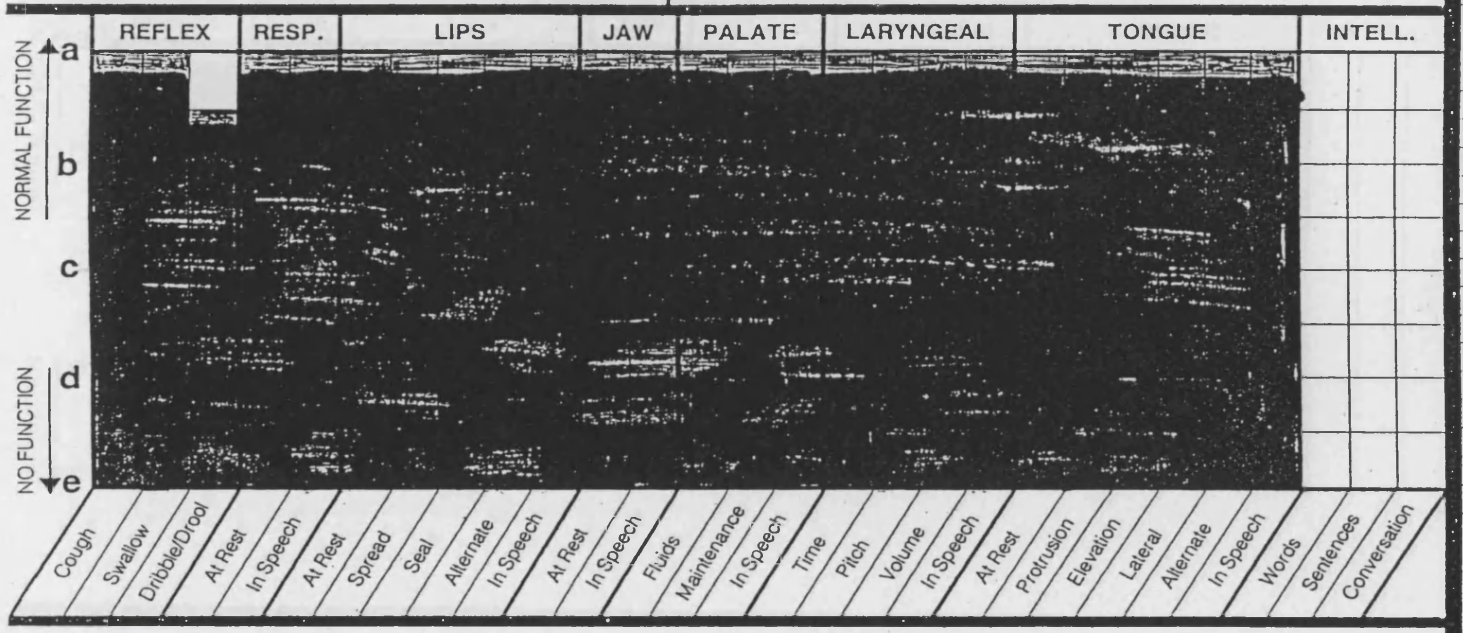
SCORING FORM
SPEECH THERAPY

NELSON

NAME OF THERAPIST KK

CLIENT NUMBER PK #4 Control

DATE OF ASSESSMENT 12/6/2000



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

PERCEPTUAL DYSARTHRIA ASSESSMENT

SCORING FORM

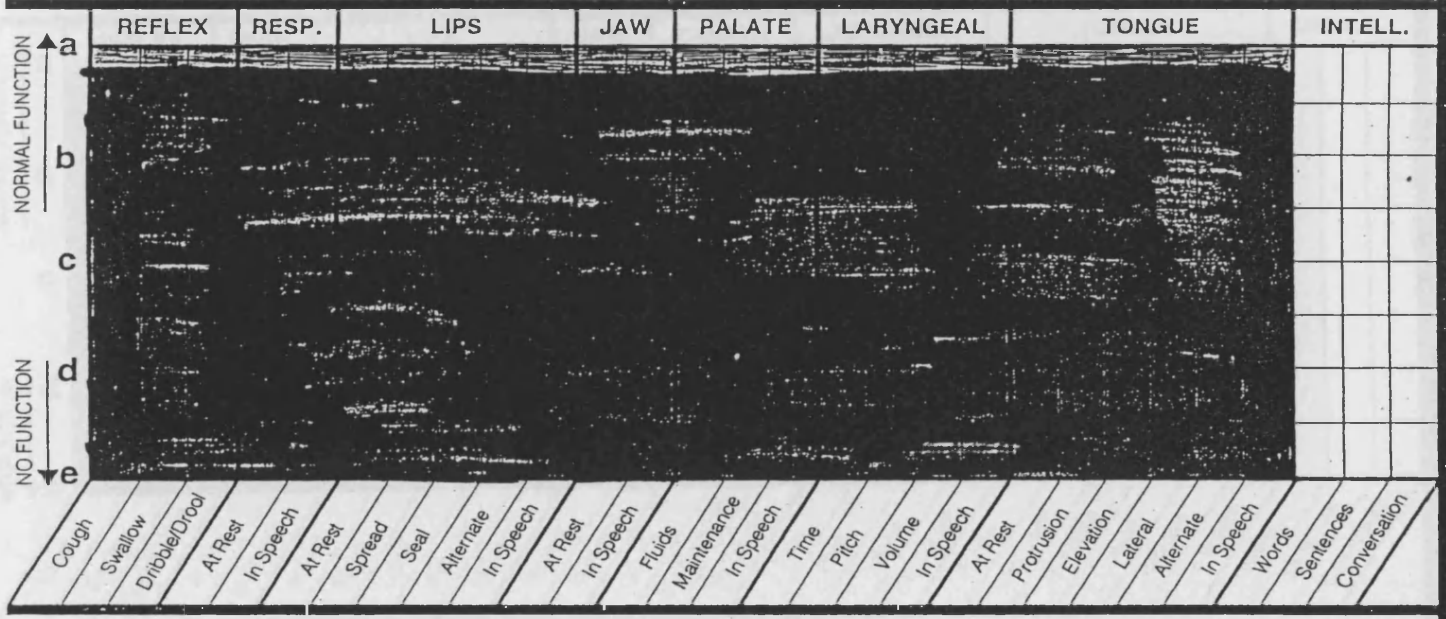
SPEECH THERAPY

NAME OF THERAPIST LK

CLIENT NUMBER THM #5 Control

DATE OF ASSESSMENT 20/2/2001

NFER NELSON



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS

Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

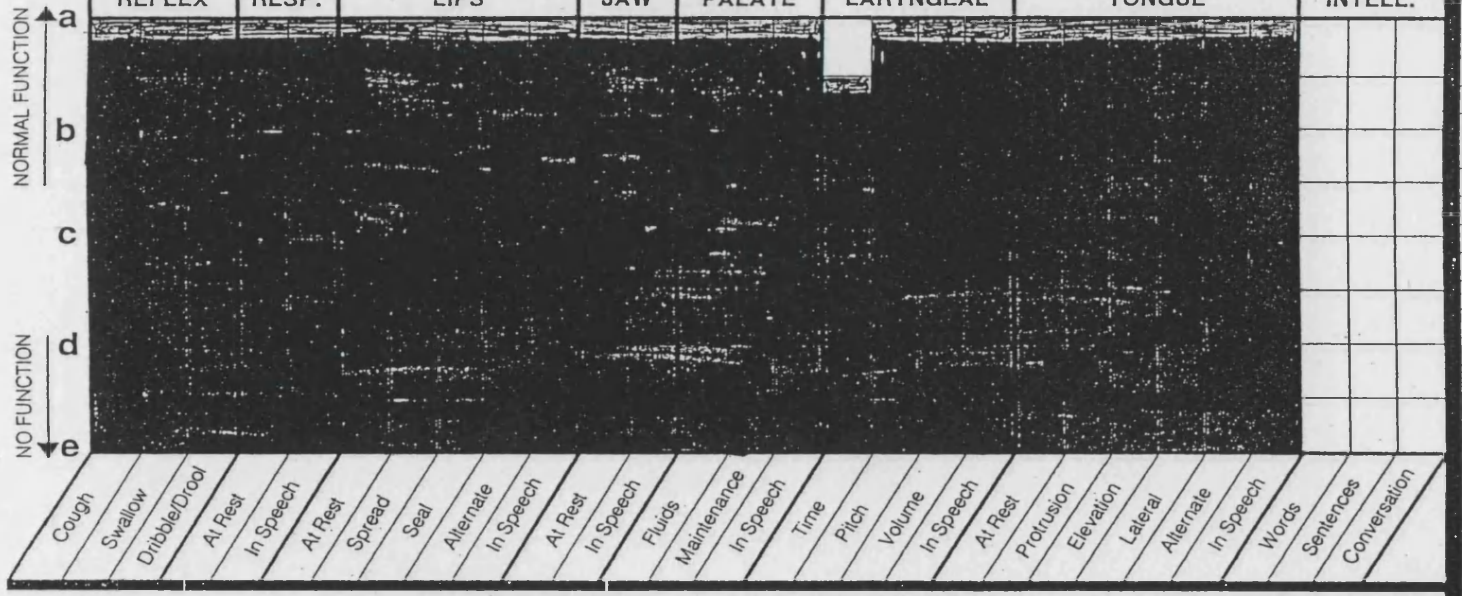
FRENCHAY DYSARTHRIA ASSESSMENT
SCORING FORM
SPEECH THERAPY

NAME OF THERAPIST KK.

CLIENT NUMBER BI #6 Control

DATE OF ASSESSMENT 13/6/2000

ELSON



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder

	×
HEARING	
SIGHT	
TEETH	
LANGUAGE	
MOOD	
POSTURE	

OTHER FACTORS

RATE (Words/Min)	
SENSATION	
UPPER LIP R	
UPPER LIP L	
TONGUE TIP	
SUBJECTIVE REPORT ON SENSATION	

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

FRENCHAY DYSARTHRIA ASSESSMENT

SCORING FORM

SPEECH THERAPY

NAME OF THERAPIST KV

CLIENT NUMBER SB #7 Control

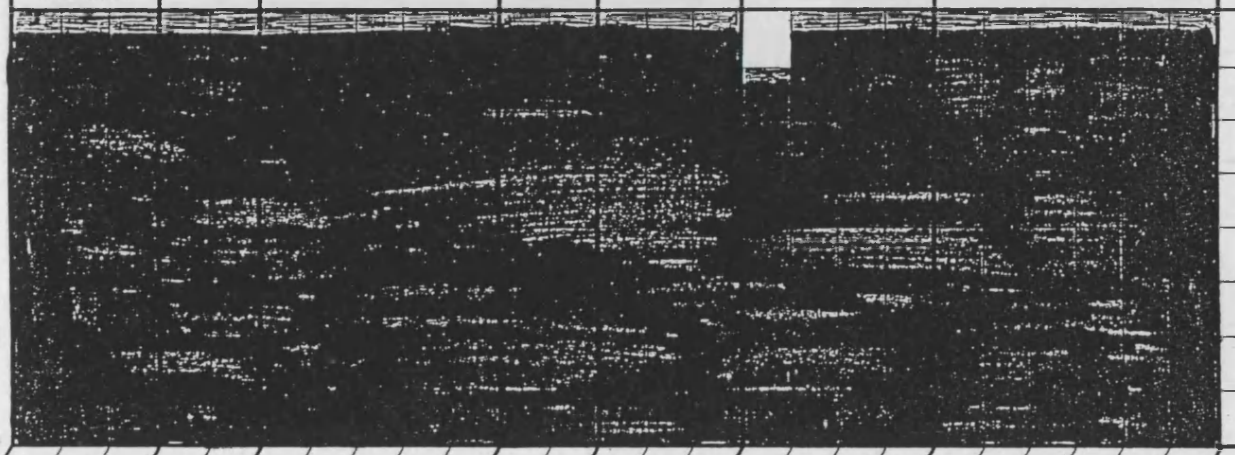
DATE OF ASSESSMENT 16/6/2000

NELSON

NORMAL FUNCTION a b c

NO FUNCTION d e

REFLEX			RESP.		LIPS			JAW		PALATE			LARYNGEAL			TONGUE					INTELL.		
Cough	Swallow	Dribble/Drool	At Rest	In Speech	At Rest	Spread	Seal	Alternate	In Speech	At Rest	In Speech	Fluids	Maintenance	In Speech	Time	Pitch	Volume	In Speech	At Rest	Protrusion	Elevation	Lateral	Alternate	In Speech	Words	Sentences	Conversation



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS

Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

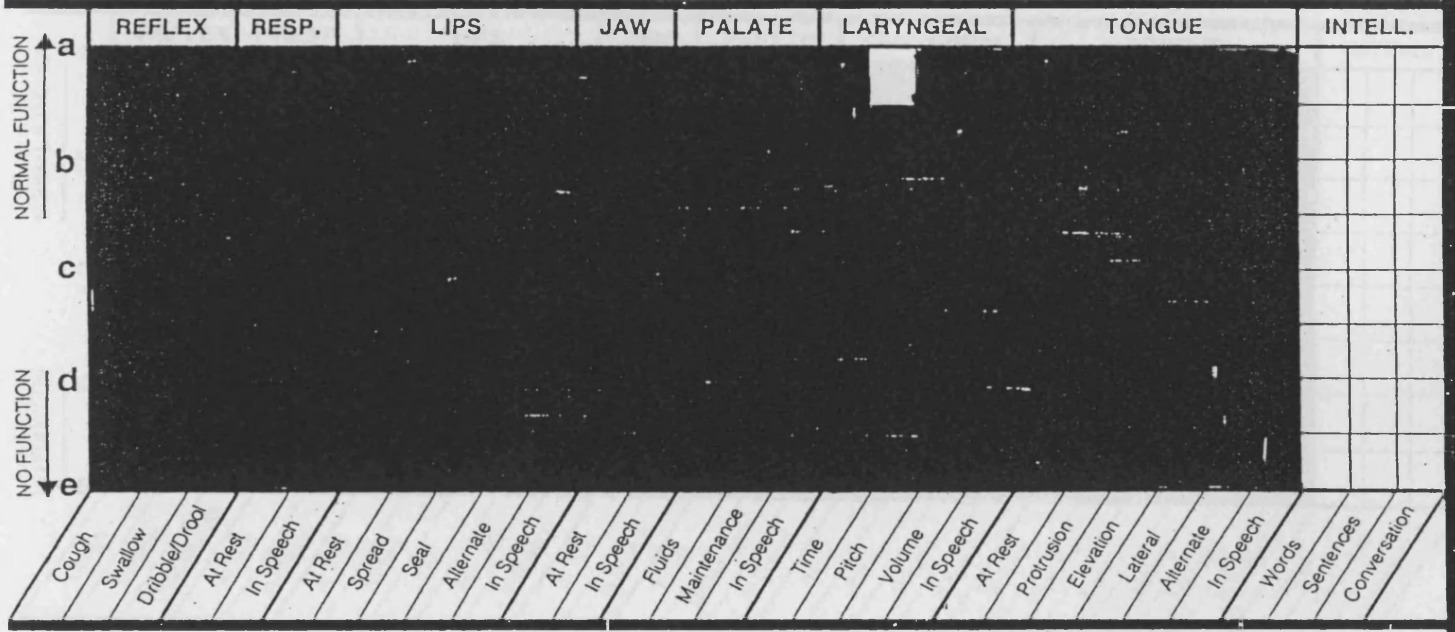
SCORING FORM
SPEECH THERAPY

NOSTE

NAME OF THERAPIST KK

CLIENT NUMBER AA # 8 Control

DATE OF ASSESSMENT 18/6/2000



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

FRENCHAY DYSARTHRIA ASSESSMENT

SCORING FORM

SPEECH THERAPY

NAME OF THERAPIST KK

CLIENT NUMBER KP #9 Control

DATE OF ASSESSMENT 10/2/2001

NORMAL FUNCTION

NO FUNCTION

	REFLEX	RESP.	LIPS	JAW	PALATE	LARYNGEAL	TONGUE	INTELL.
a								
b								
c								
d								
e								

Cough | Swallow | Dribble/Drool | At Rest | In Speech | At Rest | Spread | Seal | Alternate | In Speech | At Rest | In Speech | Fluids | Maintenance | In Speech | Time | Pitch | Volume | In Speech | At Rest | Protrusion | Elevation | Lateral | Alternate | In Speech | Words | Sentences | Conversation

SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS

Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

FRENCHAY DYSARTHRIA ASSESSM 11

SCORING FORM

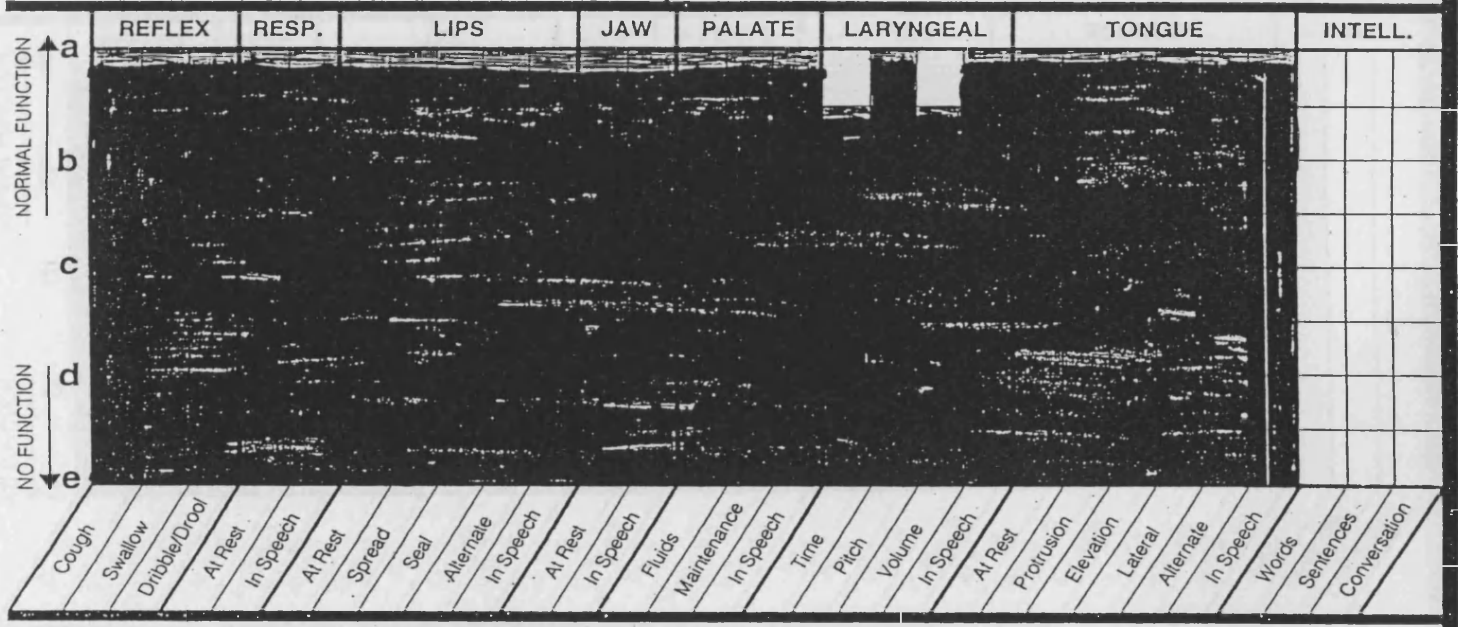
SPEECH THERAPY

NELSON

NAME OF THERAPIST KK

CLIENT NUMBER PE #10 Grp Control

DATE OF ASSESSMENT 11/7/2000



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS

Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FRENCHAY DYSARTHRIA ASSESSMENT

NELSON

SCORING FORM

SPEECH THERAPY

FAMILY NAME

FIRST NAME/S

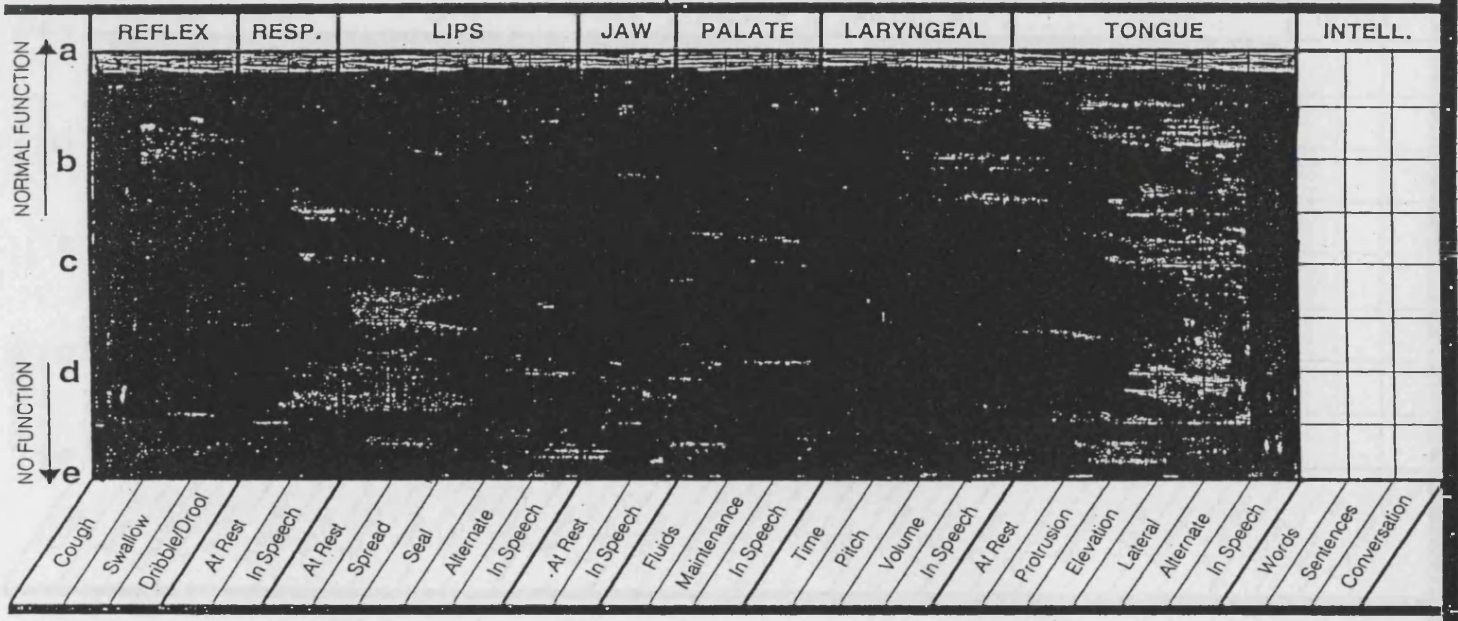
ADDRESS

DATE OF BIRTH

NAME OF THERAPIST KK

CLIENT NUMBER DN #11 : Control

DATE OF ASSESSMENT 9/11/2000



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS

Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

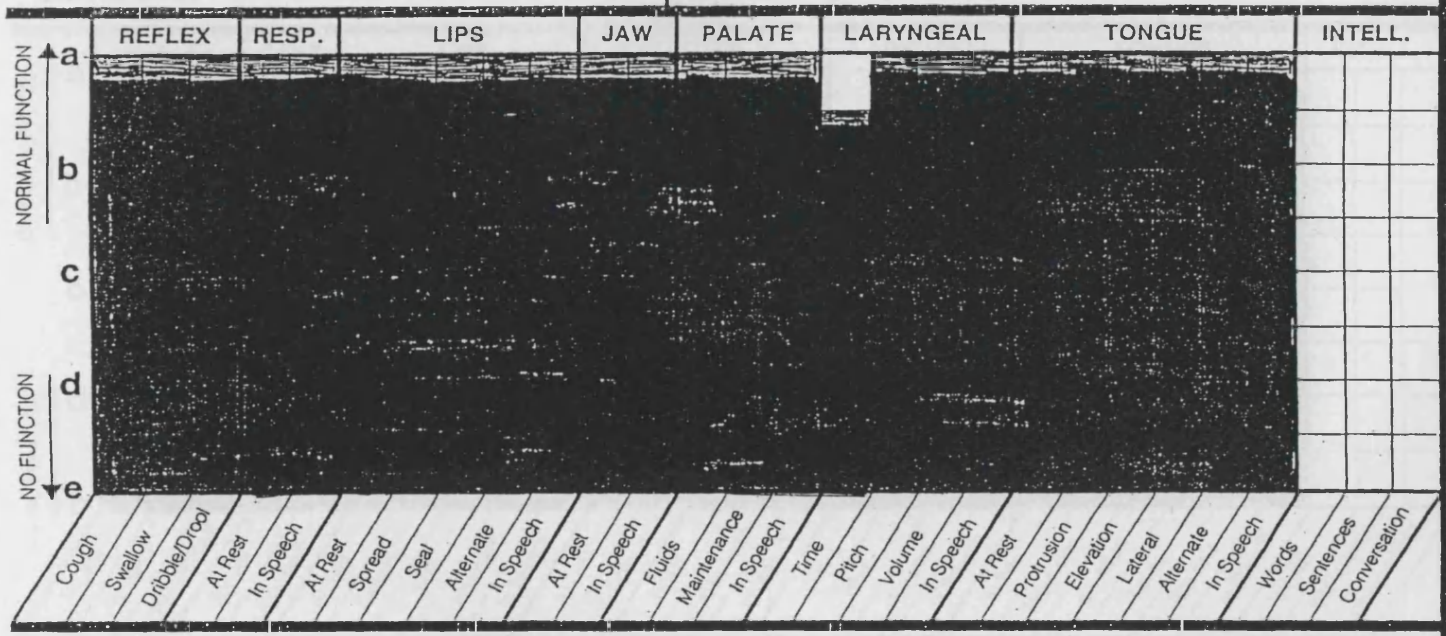
SCORING FORM
SPEECH THERAPY

NAME OF THERAPIST KK

CLIENT NUMBER NP #12 Control

DATE OF ASSESSMENT 15/11/2000

NELSON



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

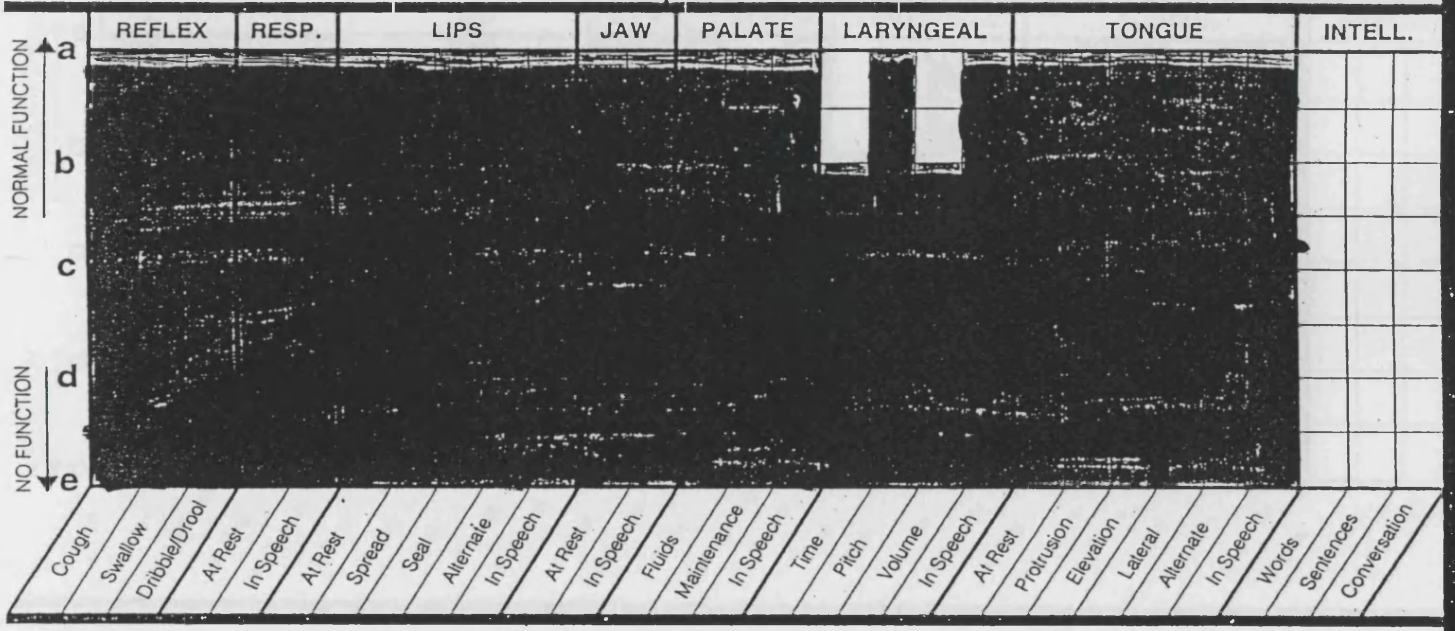
FRENCHAY DYSARTHRIA ASSESSMENT

NELSON

SCORING FORM
SPEECH THERAPY

FAMILY NAME
FIRST NAME/S
ADDRESS
DATE OF BIRTH

NAME OF THERAPIST KL
CLIENT NUMBER GD # 13 Control
DATE OF ASSESSMENT 18/11/2000



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder

HEARING
SIGHT
TEETH
LANGUAGE
MOOD
POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R
UPPER LIP L
TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

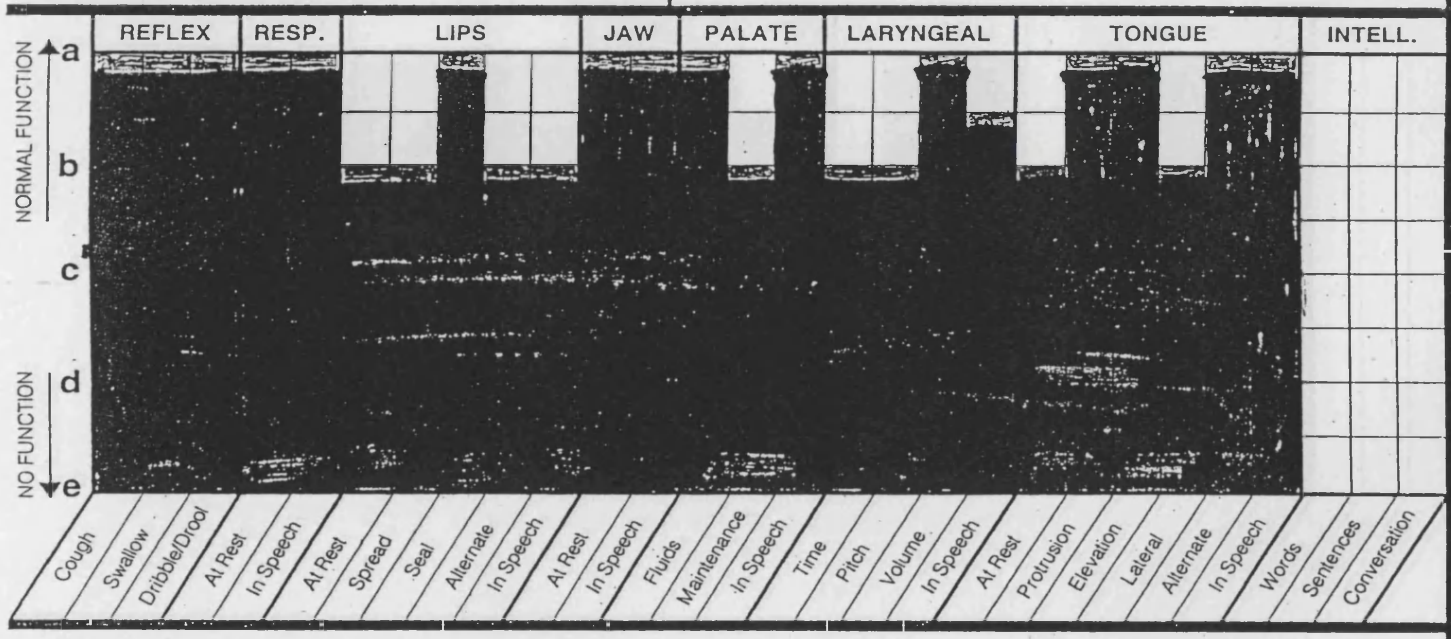
SCORING FORM
SPEECH THERAPY

NAME OF THERAPIST KK

CLIENT NUMBER TZS #2 Experimental

DATE OF ASSESSMENT 5/11/99 Before medication -

NELSON



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

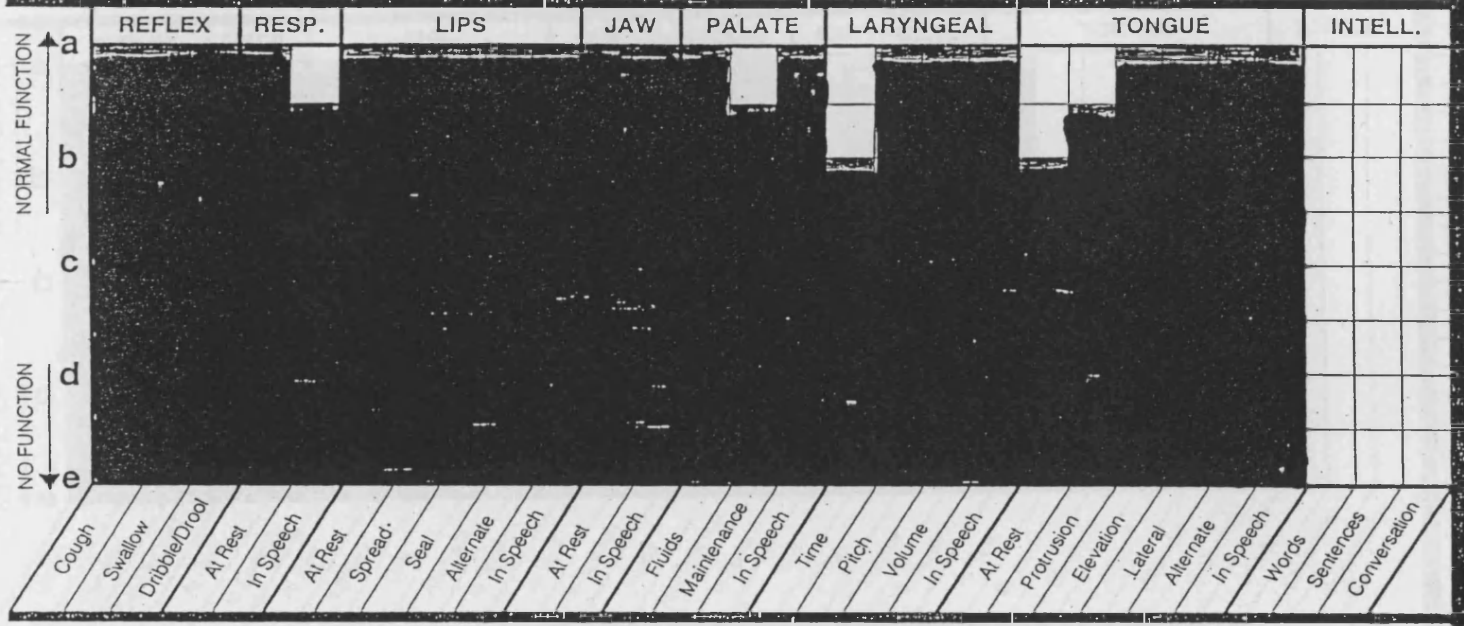
SCORING FORM
SPEECH THERAPY

NAME OF THERAPIST K.K

CLIENT NUMBER TZS#2 Experimental

DATE OF ASSESSMENT 18/2/2000 After medication

NELSON



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FRENCHAY DYSARTHRIA ASSESSMENT

SCORING FORM

SPEECH THERAPY

NELSON

FAMILY NAME

FIRST NAME/S

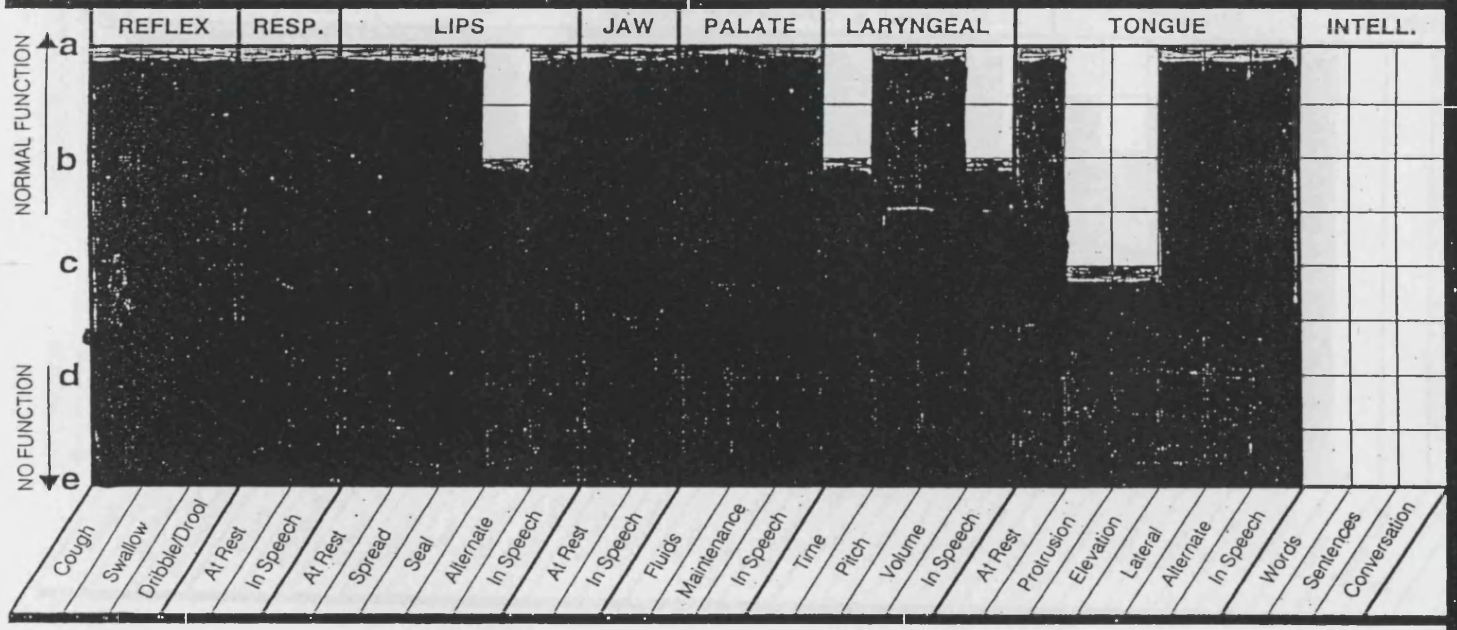
ADDRESS

DATE OF BIRTH

NAME OF THERAPIST KK

CLIENT NUMBER KD #4 experimental

DATE OF ASSESSMENT 26/11/1999 Before medication



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS

Cross if contributing to speech disorder ×

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

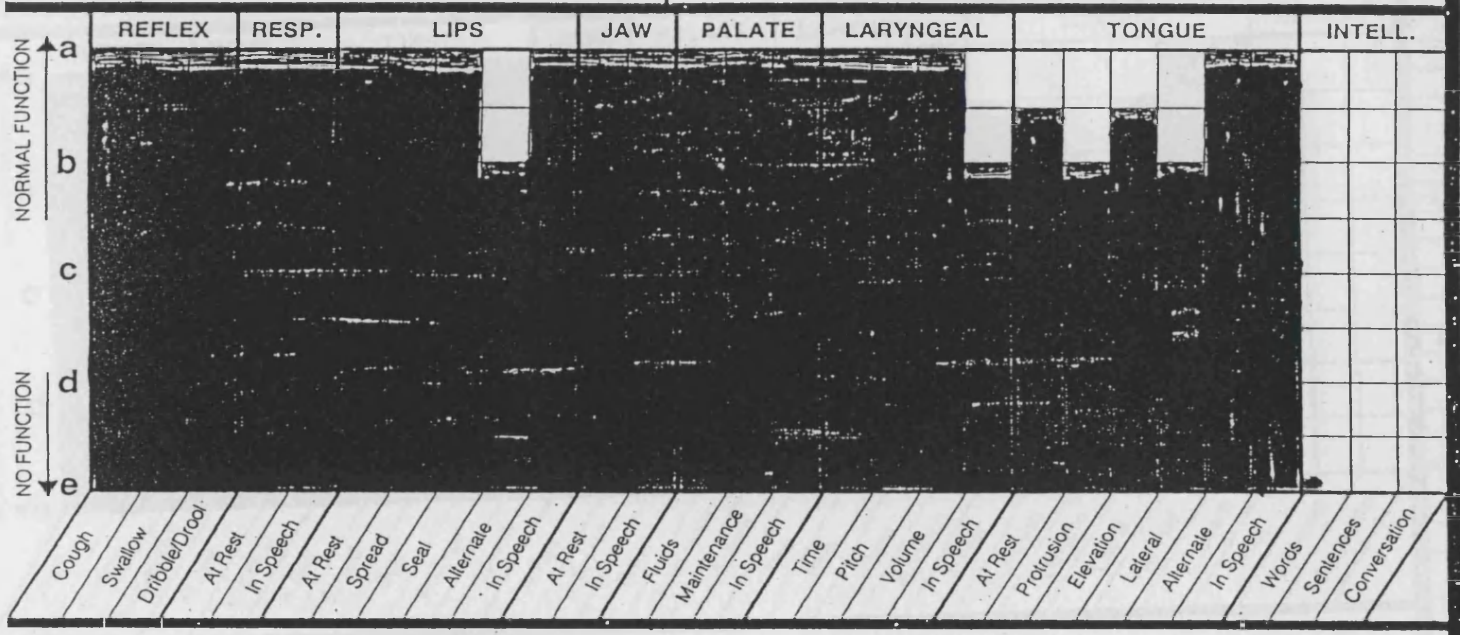
SCORING FORM
SPEECH THERAPY

NAME OF THERAPIST KK

CLIENT NUMBER KD#4 Experimental

DATE OF ASSESSMENT 16/3/2000 After medication

NELSON



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

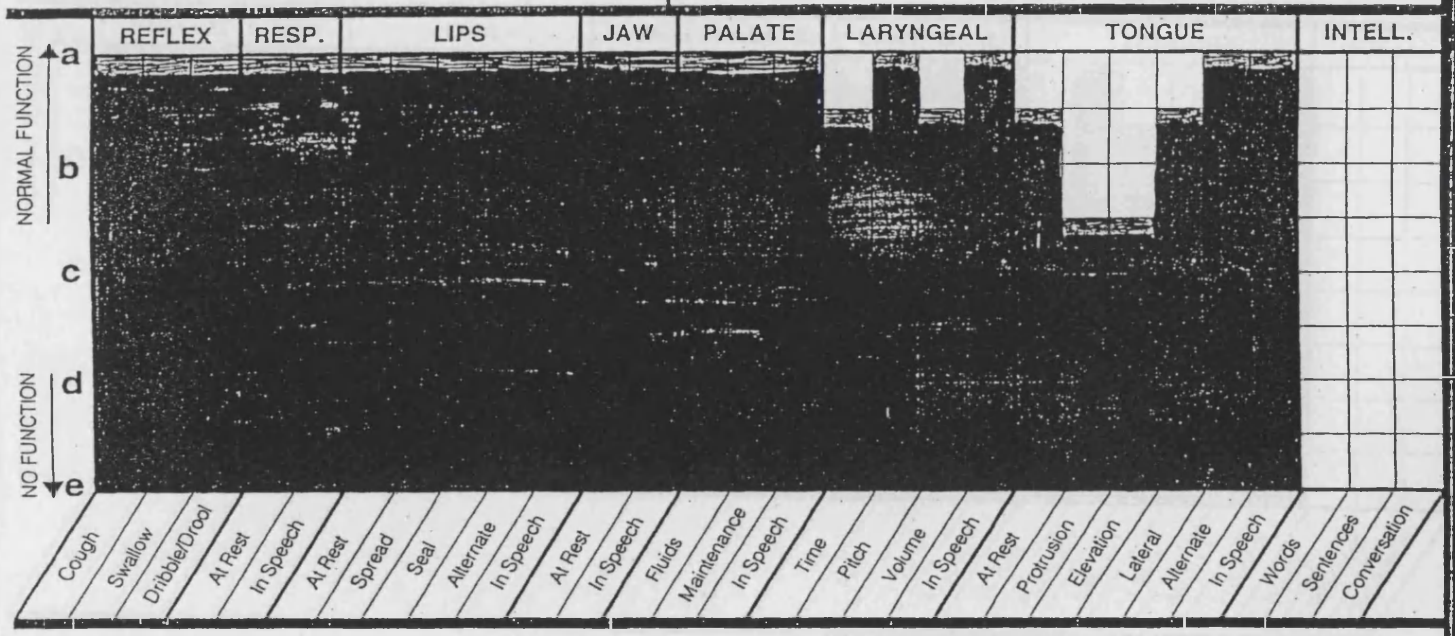
SCORING FORM
SPEECH THERAPY

NAME OF THERAPIST K K

CLIENT NUMBER SBS #5 Experimental

DATE OF ASSESSMENT 3/12/99 Before mediation

NOSOS



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

1/3 88) Code: 18C

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

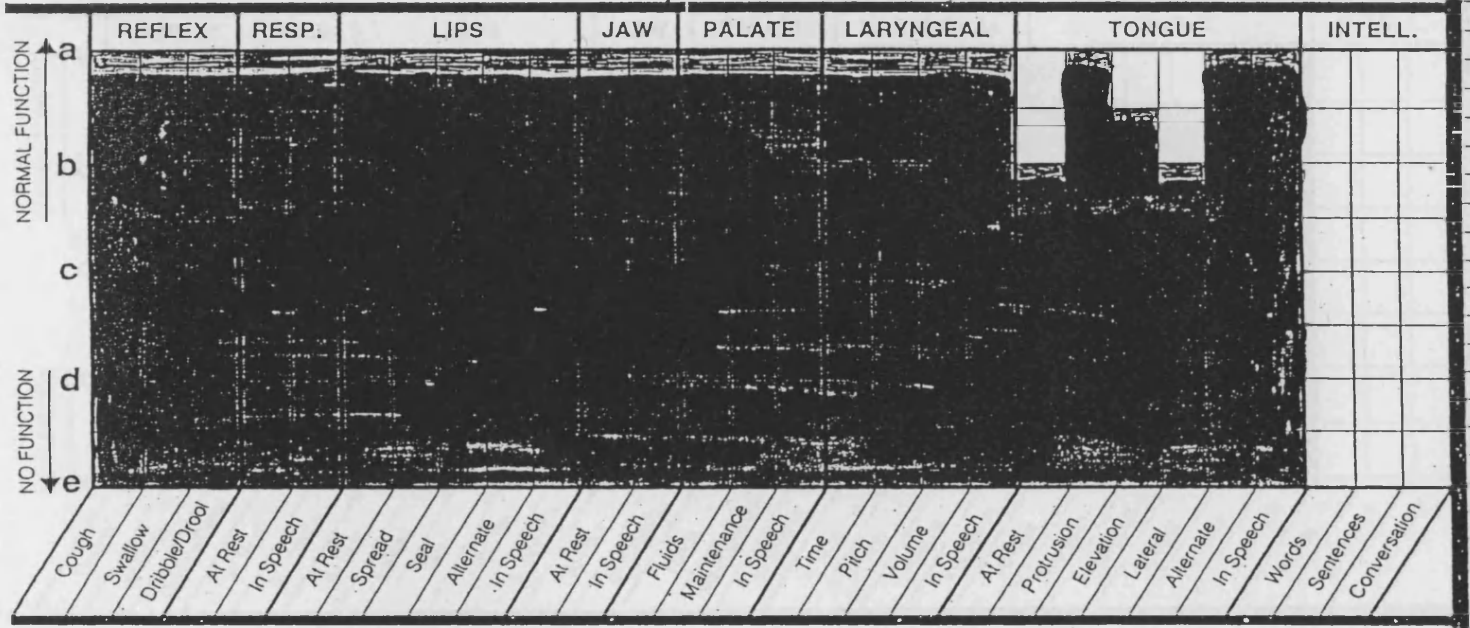
SCORING FORM
SPEECH THERAPY

NAME OF THERAPIST KK

CLIENT NUMBER SB#5 Experimental

DATE OF ASSESSMENT 31/3/2000 After medication

NOSTE



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

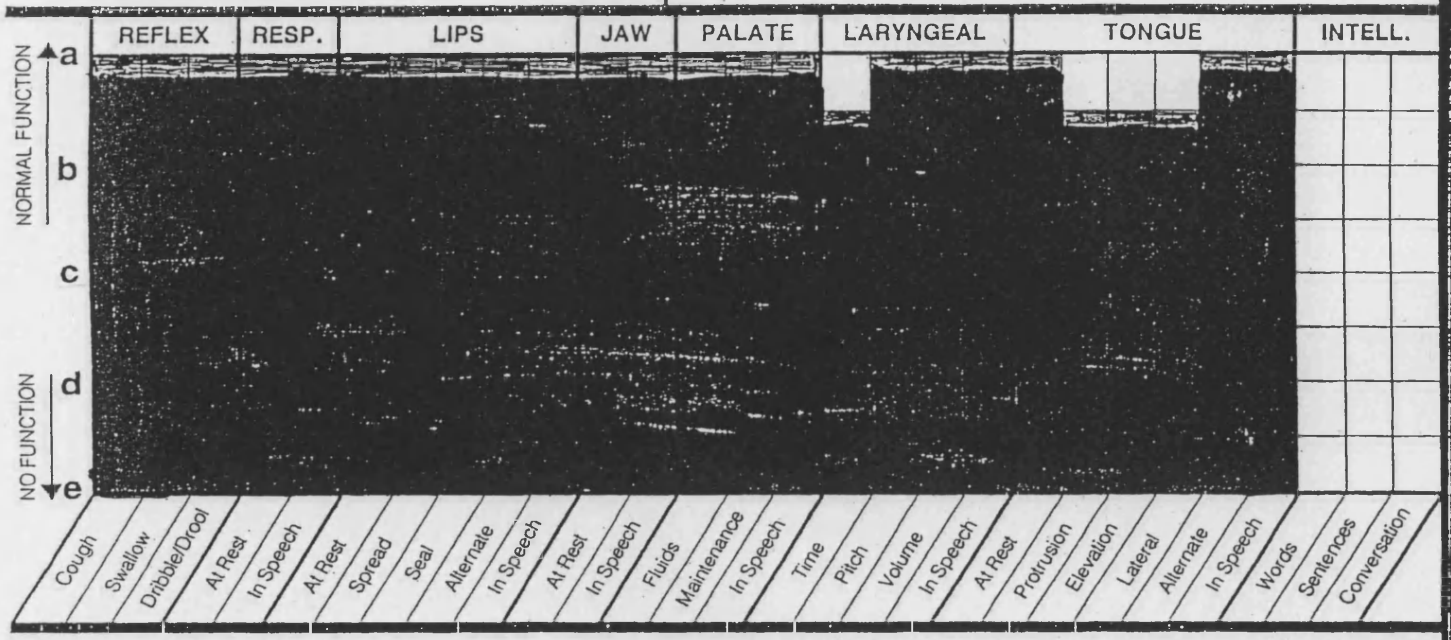
SCORING FORM
SPEECH THERAPY

NAME OF THERAPIST K-K

CLIENT NUMBER KP # 6 Experimental

DATE OF ASSESSMENT 22/12/99 Before radiation

NEL SON



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

SCORING FORM
SPEECH THERAPY

NAME OF THERAPIST KK

CLIENT NUMBER KP#6 Experimental

DATE OF ASSESSMENT 6/4/2000 After medication

NELSON

NORMAL FUNCTION

NO FUNCTION

	REFLEX	RESP.	LIPS		JAW	PALATE	LARYNGEAL	TONGUE					INTELL.		
a															
b															
c															
d															
e															
	Cough	Swallow	Dribble/Drool	At Rest	In Speech	At Rest	Spread	Seal	Alternate	In Speech	At Rest	In Speech	Fluids	Maintenance	In Speech	Time	Pitch	Volume	In Speech	At Rest	Protrusion	Elevation	Lateral	Alternate	In Speech	Words	Sentences	Conversation

SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

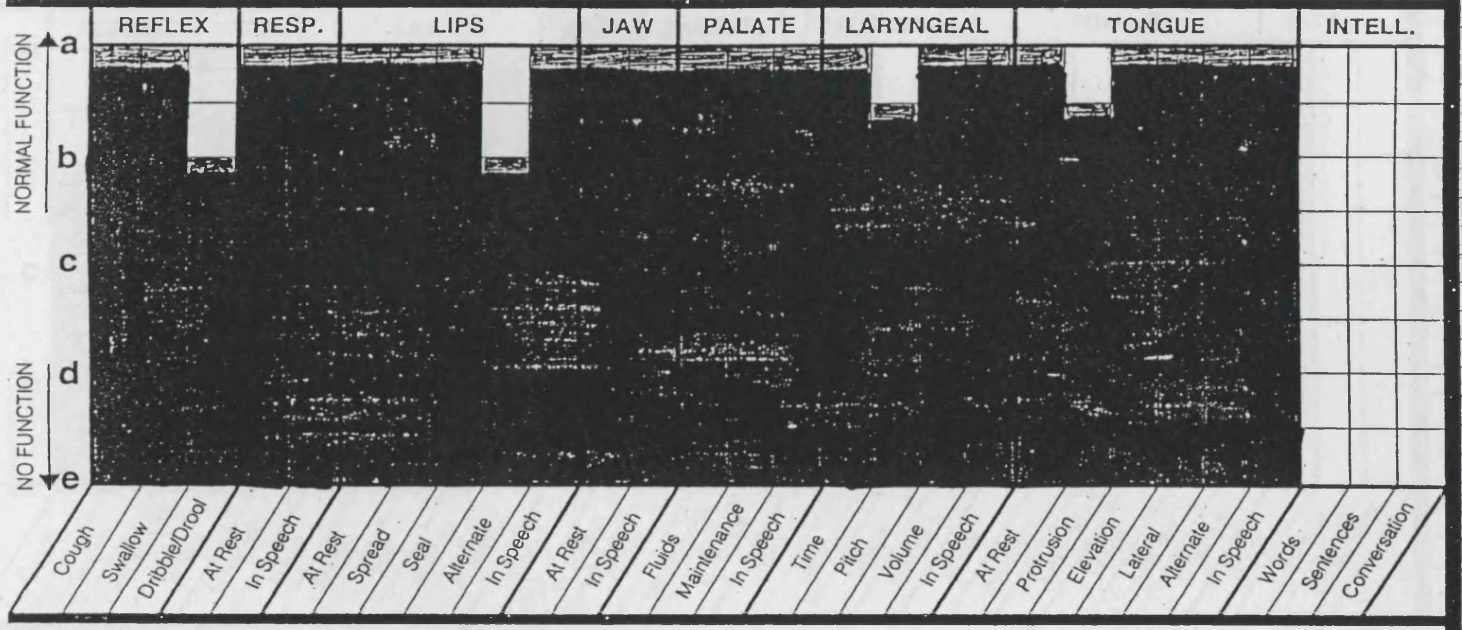
SCORING FORM
SPEECH THERAPY

NAME OF THERAPIST KK

CLIENT NUMBER PM #7 Experimental

DATE OF ASSESSMENT 14/1/2000 Before mediation

NOSTE



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

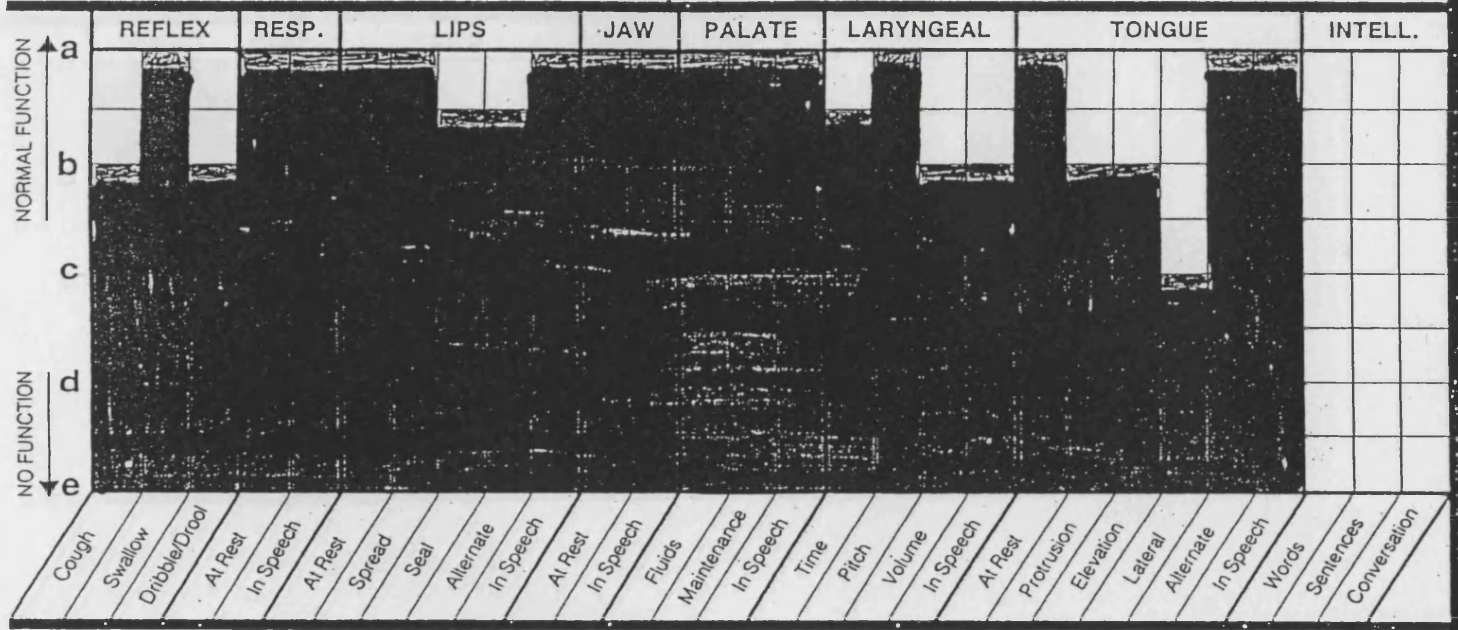
SCORING FORM
SPEECH THERAPY

NAME OF THERAPIST KK

CLIENT NUMBER PM#7 Experimental

DATE OF ASSESSMENT 1/5/2000 After medication

NOSTE



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder

HEARING	
SIGHT	
TEETH	
LANGUAGE	
MOOD	
POSTURE	

OTHER FACTORS

RATE (Words/Min)	
SENSATION	
UPPER LIP R	
UPPER LIP L	
TONGUE TIP	
SUBJECTIVE REPORT ON SENSATION	
SIGNED	

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

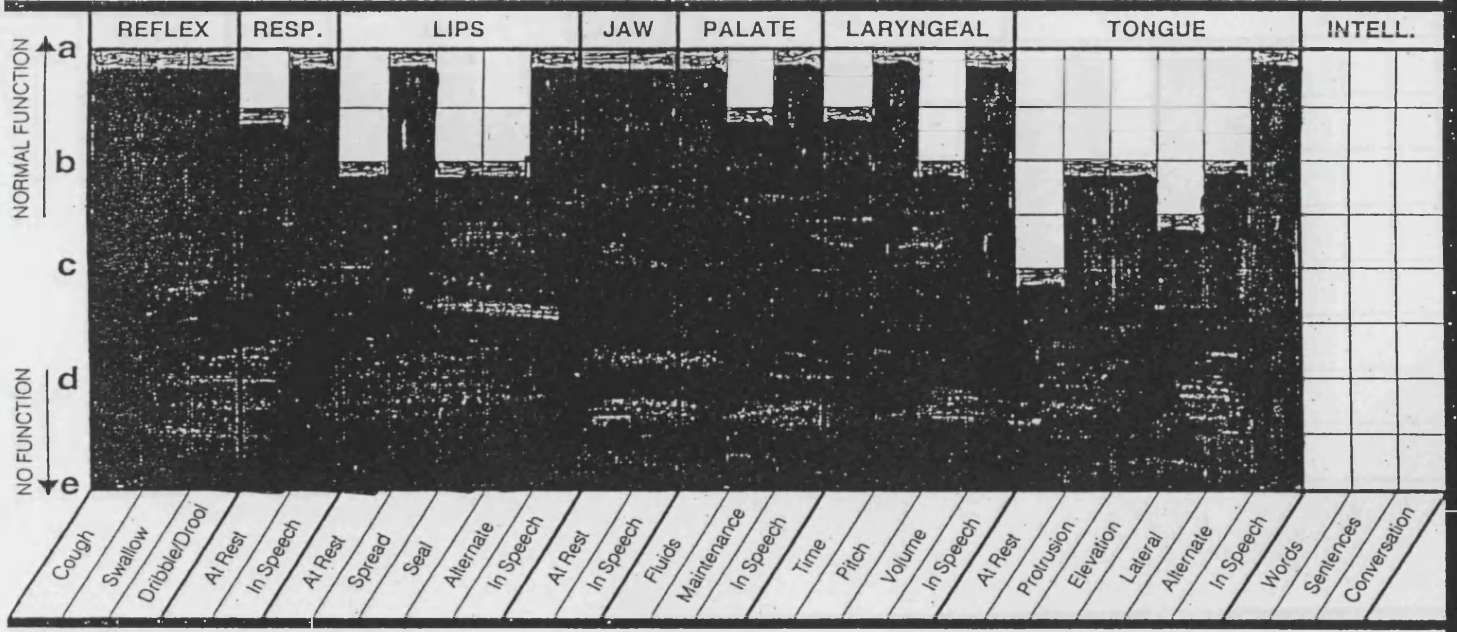
SCORING FORM
SPEECH THERAPY

NAME OF THERAPIST ICK

CLIENT NUMBER B1 #8 Experimental

DATE OF ASSESSMENT 21/4/2000 Before medication

NOSIE



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder ×

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

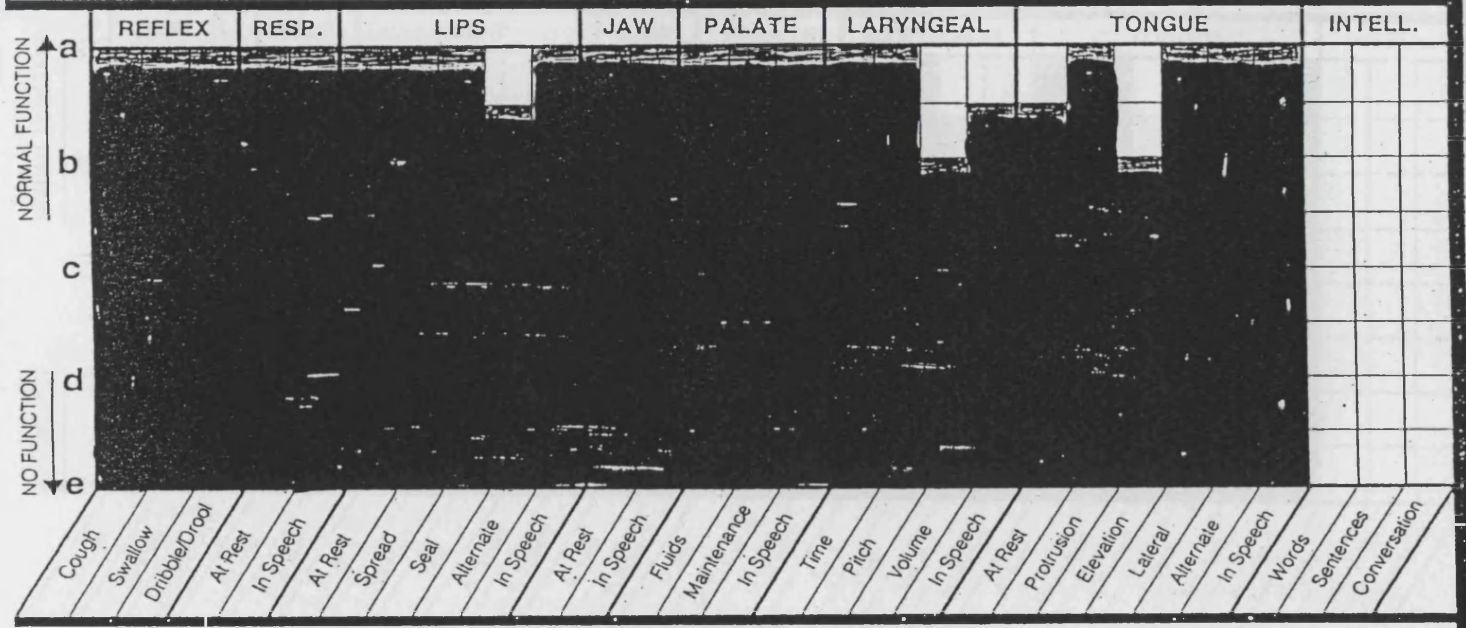
DATE OF BIRTH

SCORING FORM
SPEECH THERAPY

NAME OF THERAPIST K IC

CLIENT NUMBER B1 #8 Experimental

DATE OF ASSESSMENT 24/7/2000 After mediation



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

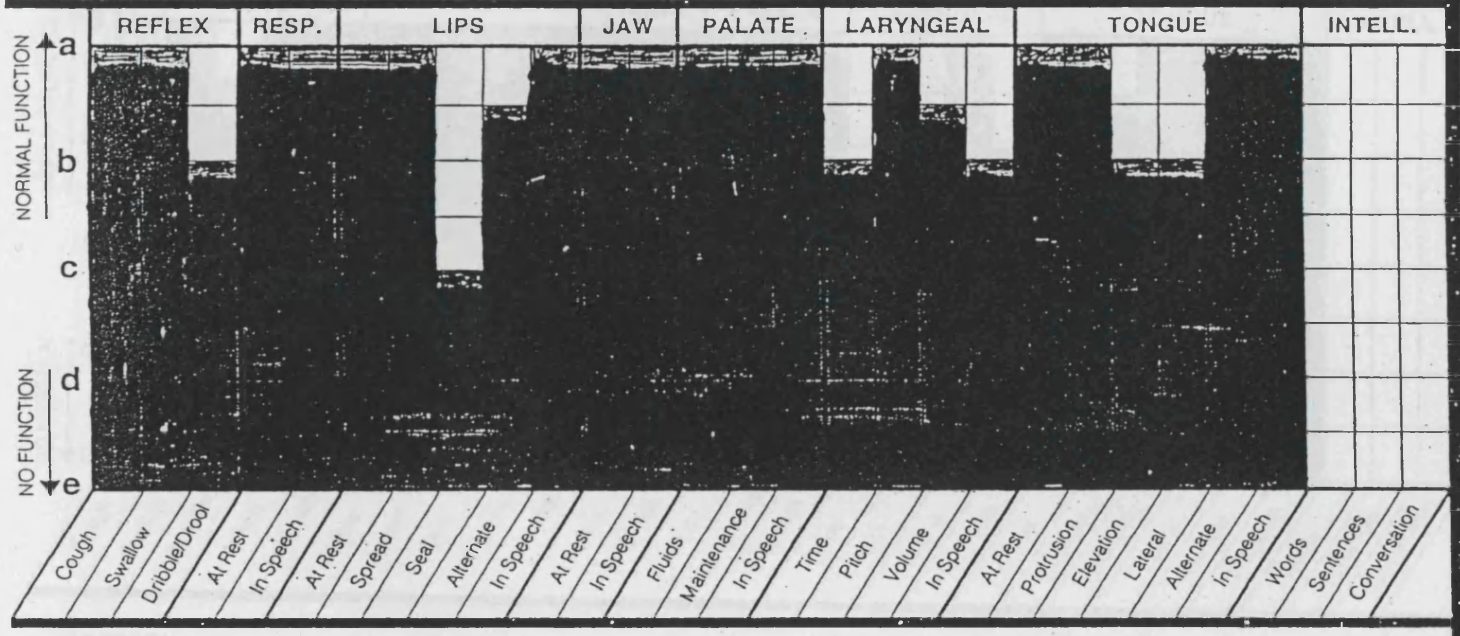
DATE OF BIRTH

SCORING FORM
SPEECH THERAPY

NAME OF THERAPIST KL

CLIENT NUMBER PX#9 'Experimental

DATE OF ASSESSMENT 30/5/2000 Before medication



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

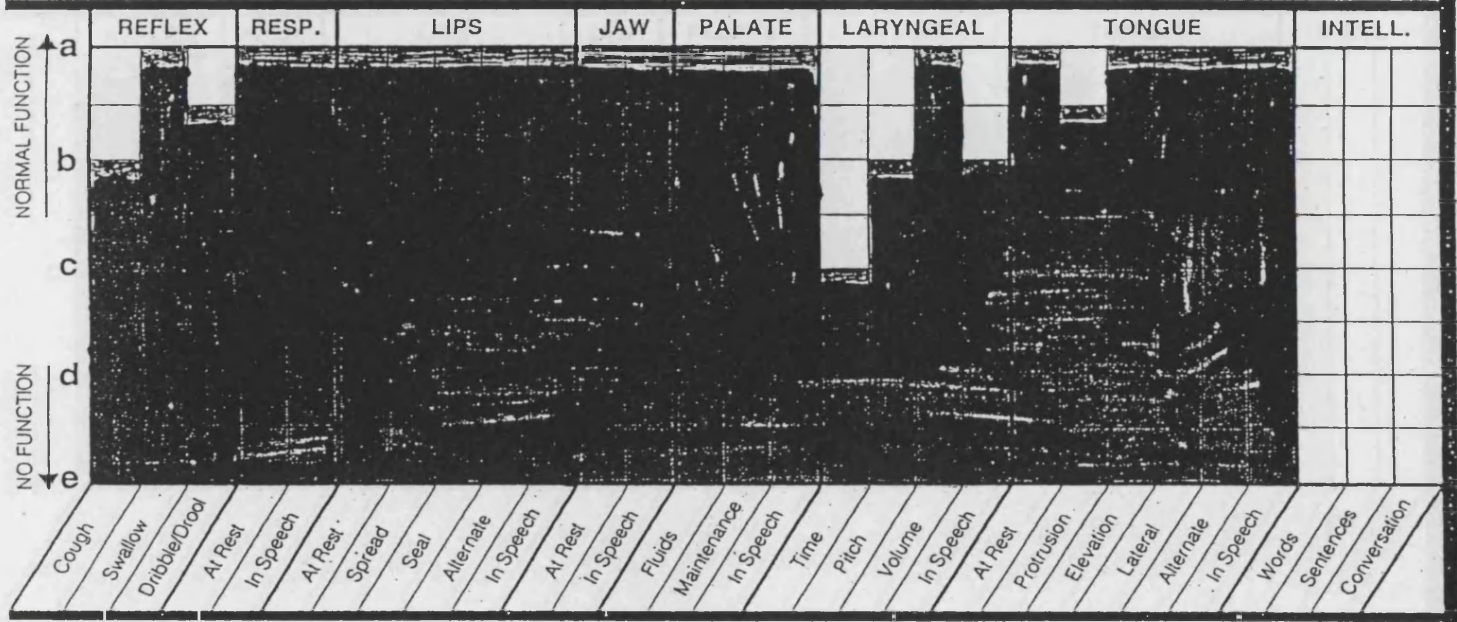
SCORING FORM
SPEECH THERAPY

NELSON

NAME OF THERAPIST KK

CLIENT NUMBER Px #9 Experimental

DATE OF ASSESSMENT 15/9/2000 After medication



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

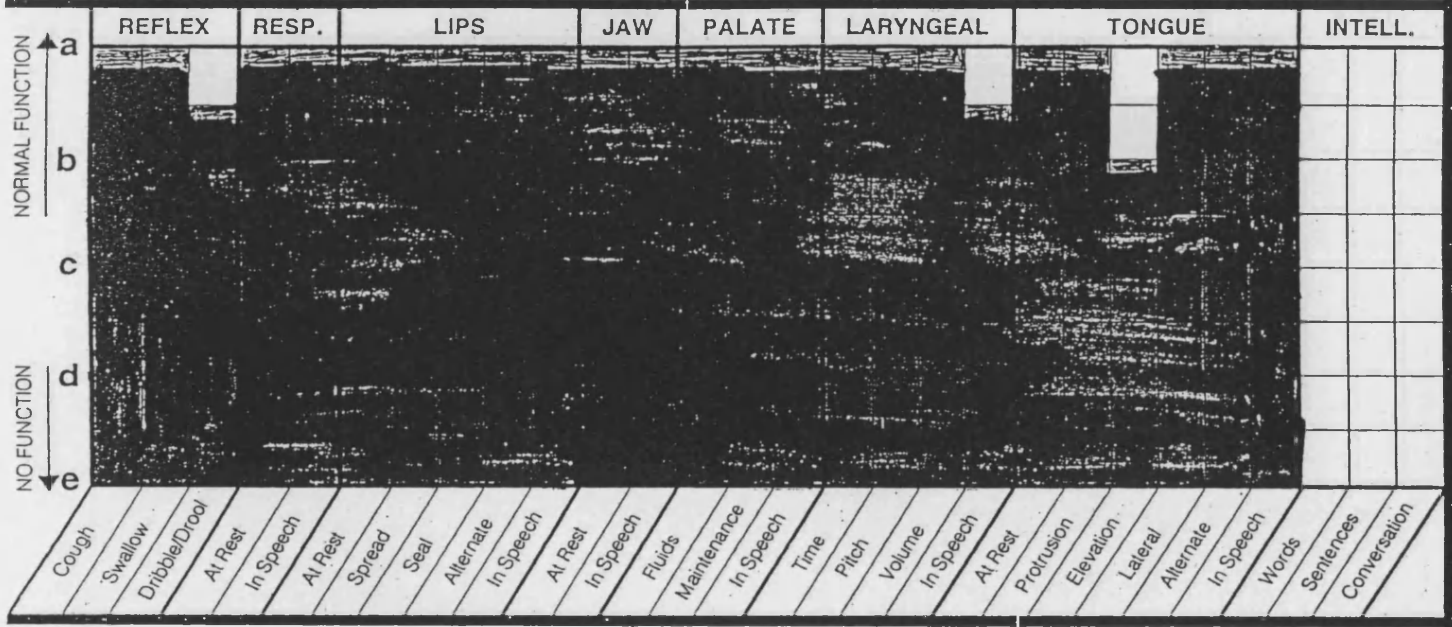
SCORING FORM
SPEECH THERAPY

NAME OF THERAPIST KK

CLIENT NUMBER SI #10 Experimental

DATE OF ASSESSMENT 1/6/2000 Before vaccination

NELSON



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder ×

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

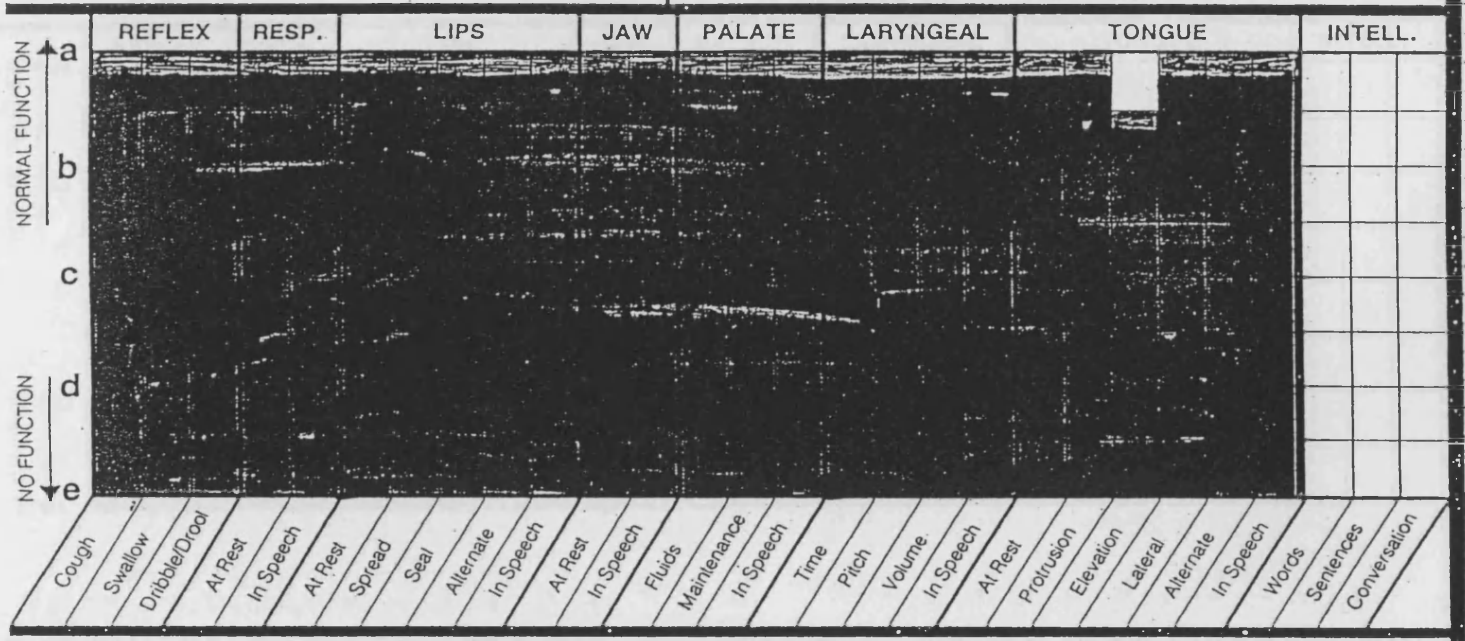
SCORING FORM
SPEECH THERAPY

NAME OF THERAPIST KK

CLIENT NUMBER S1 #10 Experimental

DATE OF ASSESSMENT 15/9/2000 After medication

NOST



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

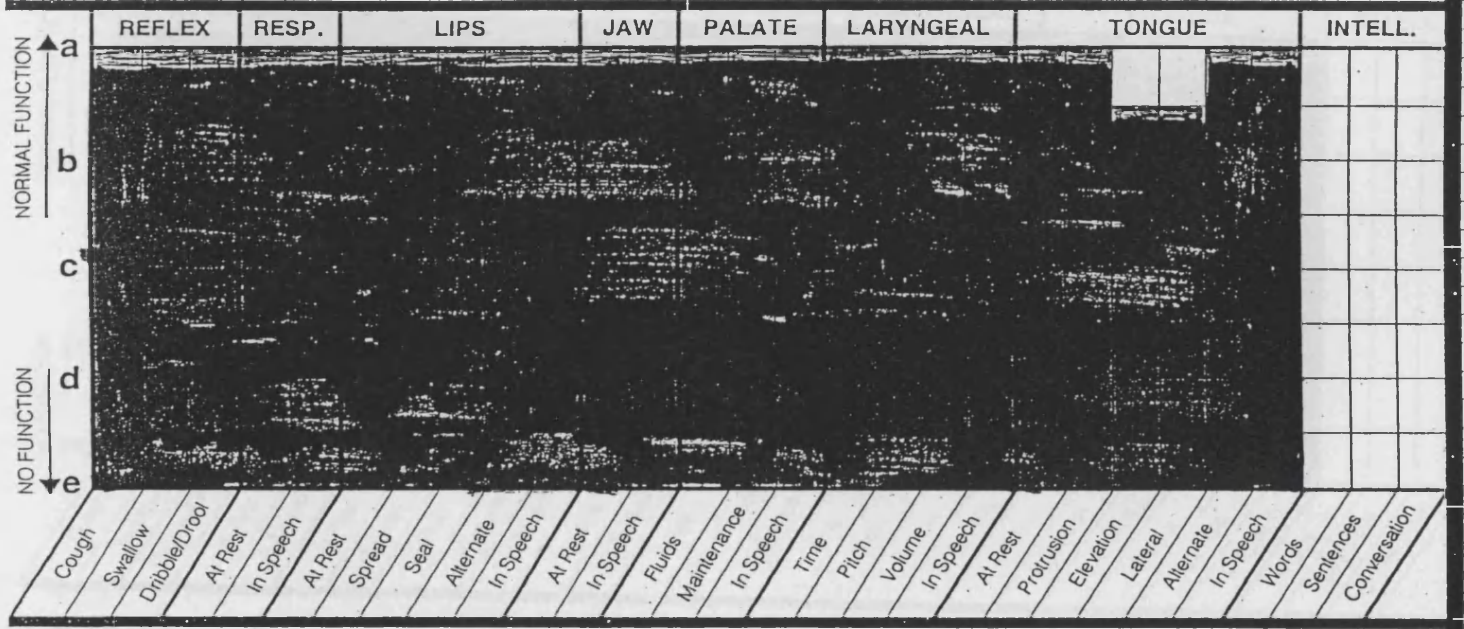
SCORING FORM
SPEECH THERAPY

NAME OF THERAPIST KL

CLIENT NUMBER GN #11 Experimental

DATE OF ASSESSMENT 8/6/2000 Before medication

NELSON



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FIRST NAME/S

ADDRESS

DATE OF BIRTH

SCORING FORM
SPEECH THERAPY

NAME OF THERAPIST KL

CLIENT NUMBER GN11 - Experimental

DATE OF ASSESSMENT 17/9/2000 After radiation

NFER-NELSON

NORMAL FUNCTION

a

b

c

NO FUNCTION

d

e

REFLEX			RESP.		LIPS			JAW		PALATE			LARYNGEAL			TONGUE									INTELL.		
Cough	Swallow	Dribble/Drool	At Rest	In Speech	At Rest	Spread	Seal	Alternate	In Speech	At Rest	In Speech	Fluids	Maintenance	In Speech	Time	Pitch	Volume	In Speech	At Rest	Protrusion	Elevation	Lateral	Alternate	In Speech	Words	Sentences	Conversation			

SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

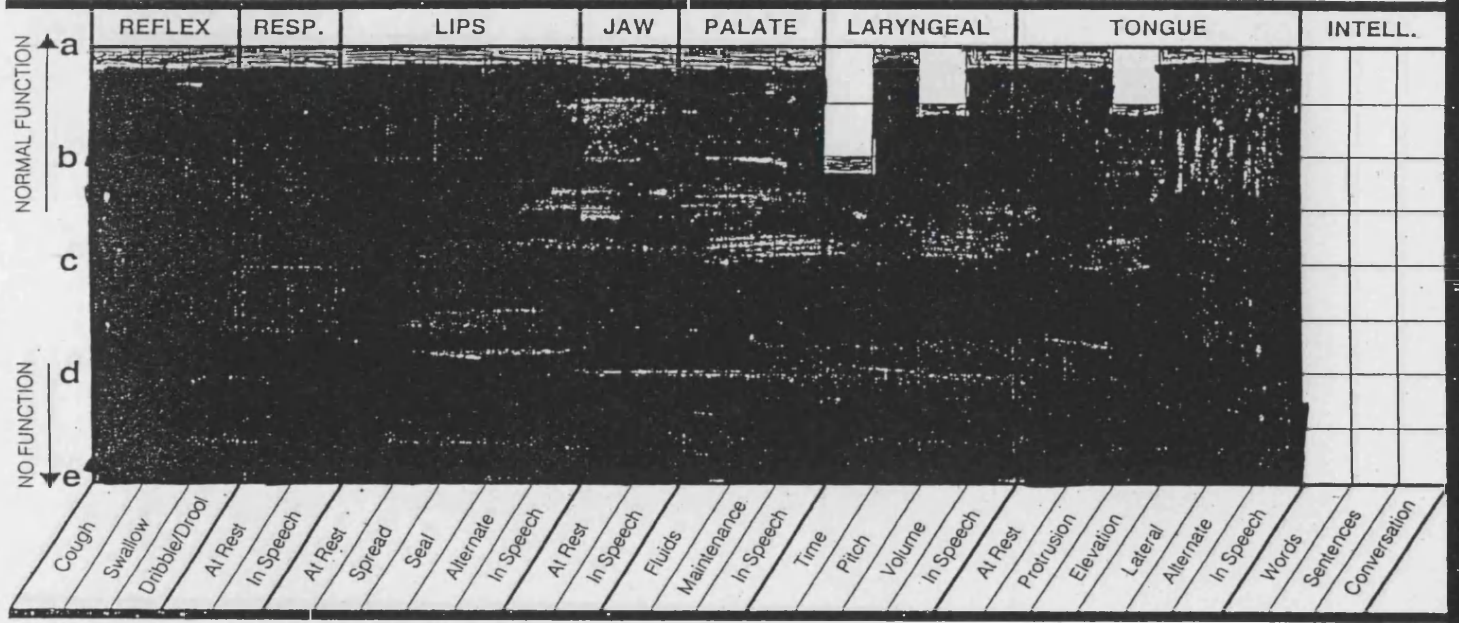
SCORING FORM
SPEECH THERAPY

NAME OF THERAPIST KK

CLIENT NUMBER GEI #13 Experimental

DATE OF ASSESSMENT 8/6/2000 Before medication

NOSTE



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder ×

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

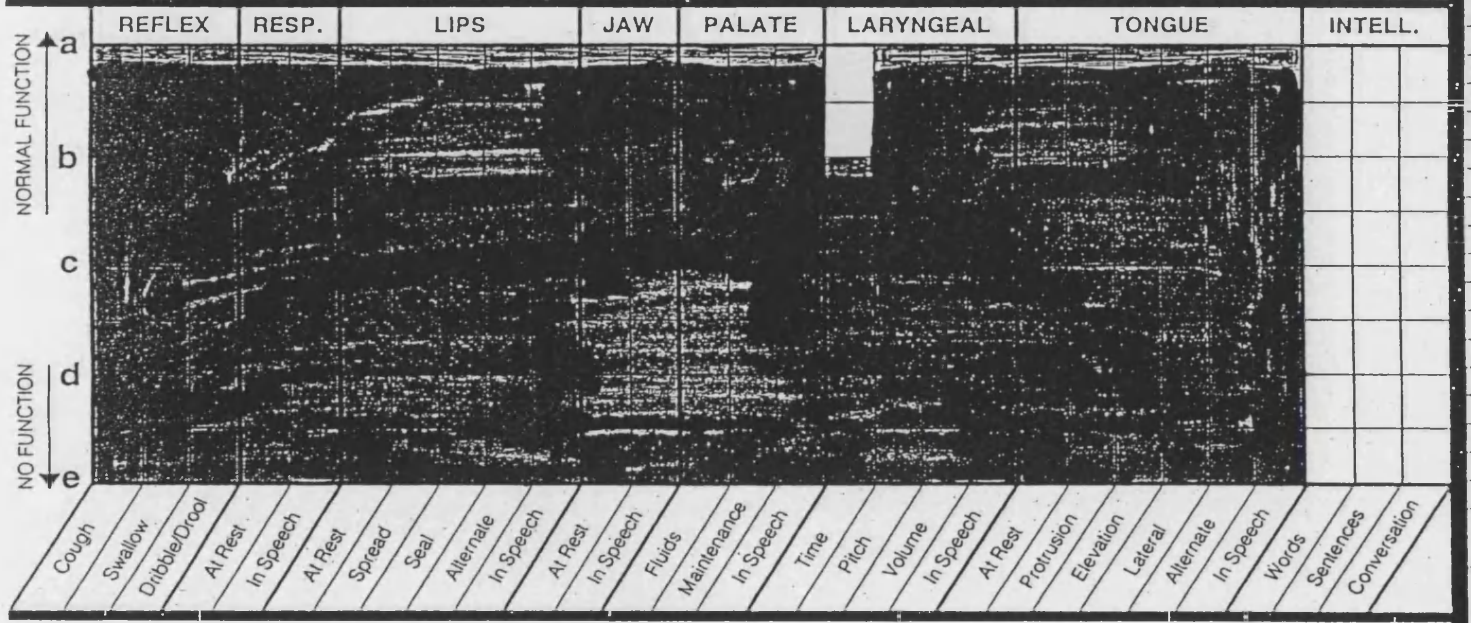
SCORING FORM
SPEECH THERAPY

NAME OF THERAPIST EK

CLIENT NUMBER GE1 #13 Experimental

DATE OF ASSESSMENT 25/10/2000 After medication

NELSON



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

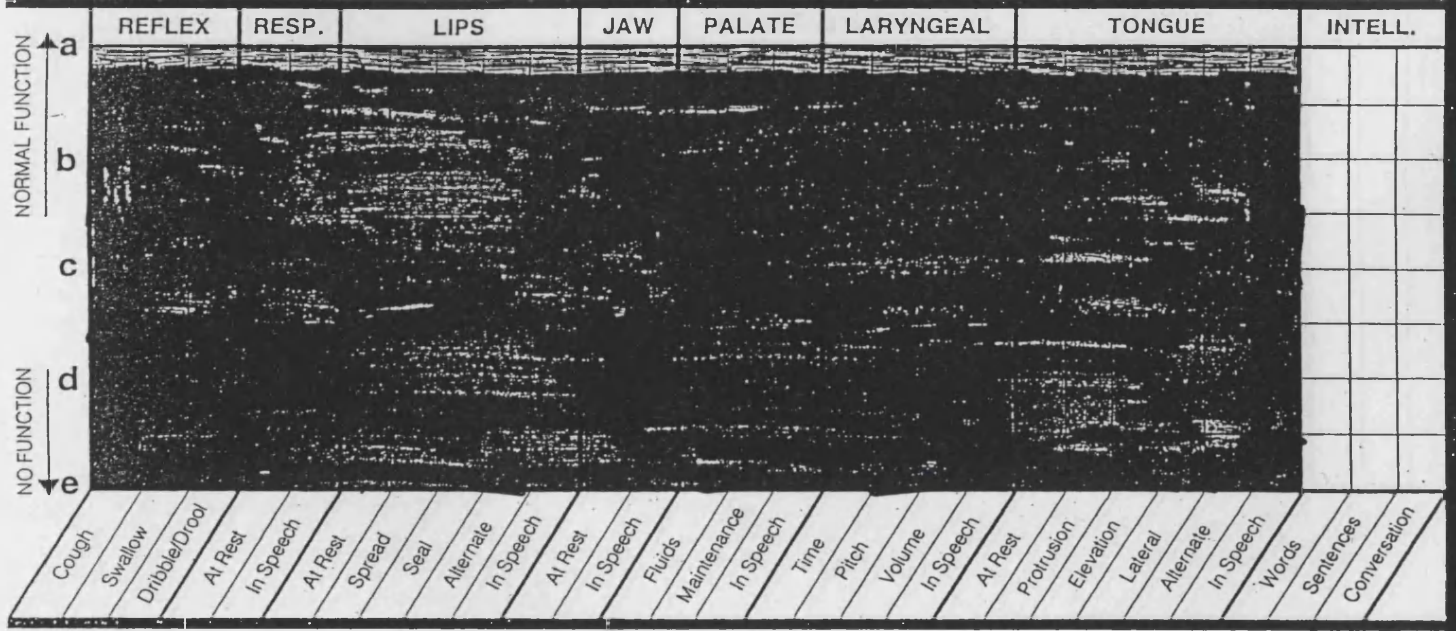
SCORING FORM
SPEECH THERAPY

NAME OF THERAPIST K-K

CLIENT NUMBER L1 #14 Experimental

DATE OF ASSESSMENT 16/6/2000 Before medication

NFER-NELSON



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

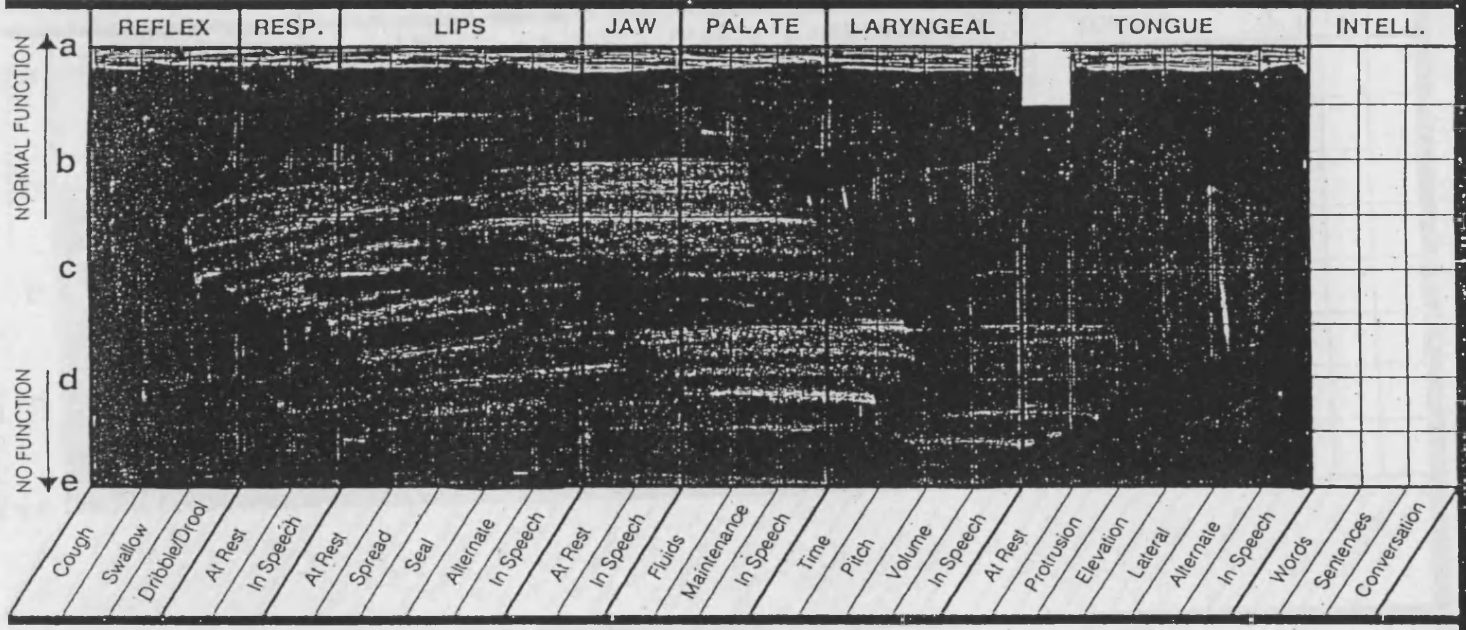
SCORING FORM
SPEECH THERAPY

NAME OF THERAPIST KK

CLIENT NUMBER CL#14 Experimental

DATE OF ASSESSMENT 3/10/2000 After medication

NFER-NELSON



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

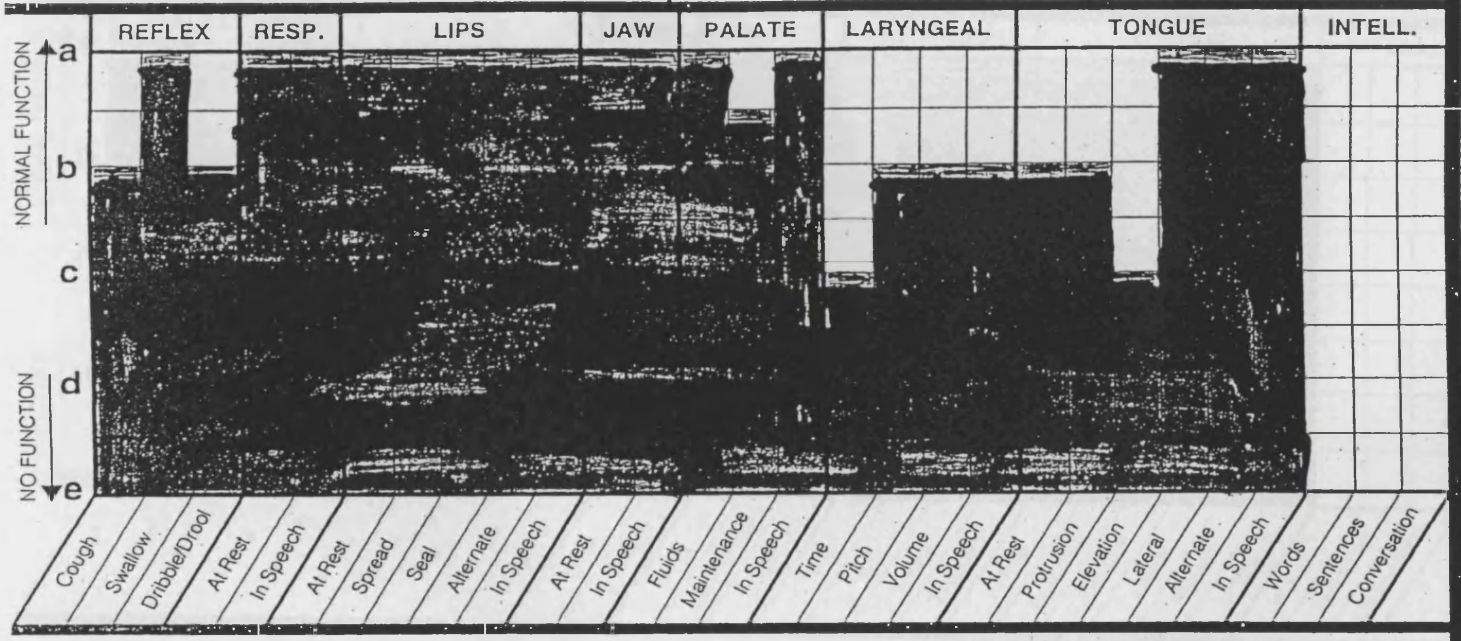
SCORING FORM
SPEECH THERAPY

NAME OF THERAPIST K-K

CLIENT NUMBER KA #15 Experimental

DATE OF ASSESSMENT 12/10/2000 Before medication

NOST



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder

	×
HEARING	
SIGHT	
TEETH	
LANGUAGE	
MOOD	
POSTURE	

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

FAMILY NAME

FIRST NAME/S

ADDRESS

DATE OF BIRTH

SCORING FORM
SPEECH THERAPY

NAME OF THERAPIST KK

CLIENT NUMBER MD # 16 Experimental

DATE OF ASSESSMENT 16/11/2000 Before motivation

NELSON

NORMAL FUNCTION

a

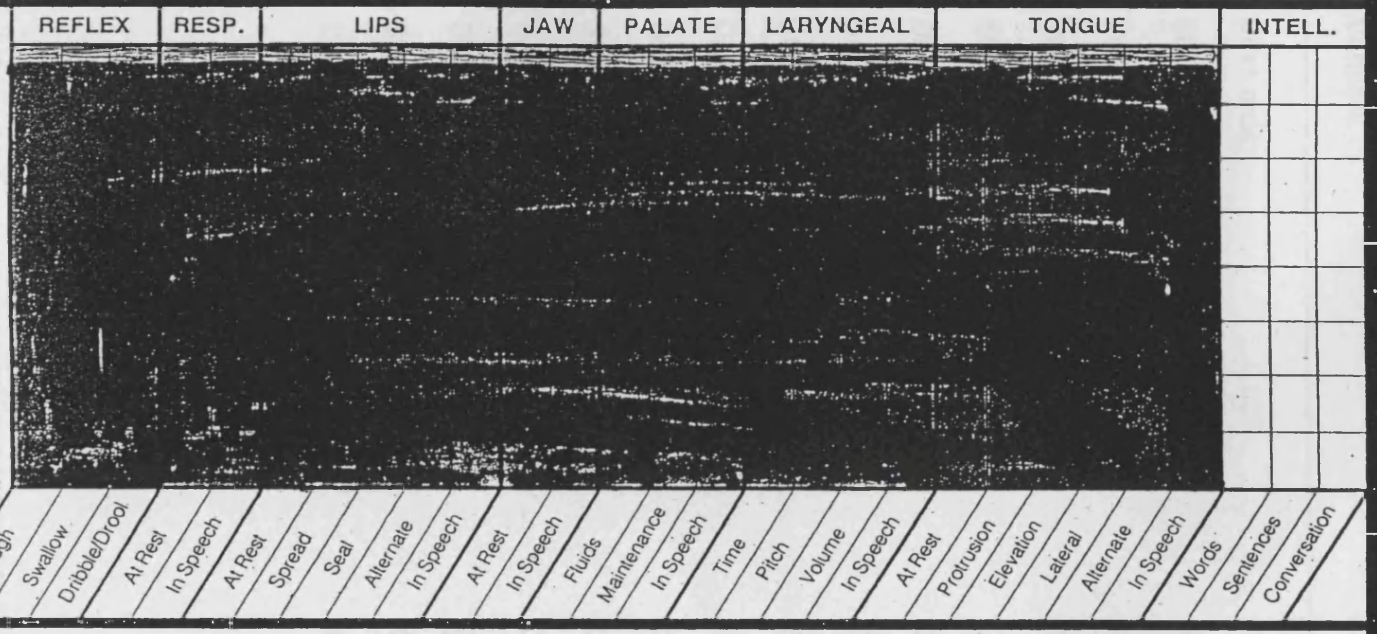
b

c

d

NO FUNCTION

e



SUMMARY

RECOMMENDATIONS

INFLUENCING FACTORS
Cross if contributing to speech disorder

HEARING

SIGHT

TEETH

LANGUAGE

MOOD

POSTURE

OTHER FACTORS

RATE (Words/Min)

SENSATION

UPPER LIP R

UPPER LIP L

TONGUE TIP

SUBJECTIVE REPORT ON SENSATION

SIGNED

Appendix O

Statistical significance between the Parkinsonian and control groups in reading compared to conversation

Within Subjects ANOVA[15]

	Parkinsonian	Control	Overall effect
	p	*p*	*p*
Variable			
DFx1 mean	0.065	0.849	0.082
SDDFx1	**0.002**	**0.008**	**0.000**
DFx1 90% range	**0.003**	**0.013**	**0.000**
DFx2 mean	0.116	0.753	0.085
SDDFx2	**0.002**	**0.043**	**0.000**
DFx2 90% range	**0.001**	**0.003**	**0.000**
DAx1 mean	**0.002**	**0.011**	**0.000**
SDDAx1	**0.028**	0.463	0.062
DAx1 90% range	**0.029**	0.418	**0.047**
DAx2 mean	**0.004**	**0.030**	**0.001**
SDDAx2	**0.007**	0.365	**0.023**
DAx2 90% range	**0.007**	0.095	**0.001**
DQx1 mean	0.834	0.179	0.364
SDDQx1	0.055	**0.000**	**0.000**
DQx1 90% range	**0.042**	**0.000**	**0.000**

[15] These results were confirmed with non parametrics (Wilcoxon) that showed the same statistical differences.

Appendix P

Tendencies of the individual scores

Subjects	DFx1 mean in conversation	DFx2 mean in conversation	DQx1 mean in conversation	DQx1 mean in reading	Average Qx in sustained phonation
BI 8	155.56	169.64	44.50	44.50	45.41
AN 2	119.96	130.81	39.50	37.50	46.53
GEI 13	201.74	207.65	35.50	37.50	35.49
THM 6	190.42	201.74	44.50	46.50	43.12
KD 4	151.13	155.56	35.50	32.50	43.57
BE 1	130.81	134.65	43.50	47.50	50.31
PM 7	110.00	110.00	45.50	45.50	38.04
KP 10	151.13	151.13	50.50	53.50	56.17
SI 10	116.54	134.65	39.50	38.50	38.84
SB 8	113.22	113.22	39.50	39.50	42.04
TZS 2	269.29	269.29	30.50	31.50	40.89
BA 4	185.00	196.00	36.50	38.50	38.40

Bold = Parkinsonian subjects

Plain = control subjects

Appendix Q

Raw scores of subject SB5 as compared to the male subgroup

READING

Subjects	DAx1 mean	SD DAx1	DAx1 Range	DAx2 mean
SB5 before	52.50	7.61	14.20	53.61
SB5 after	44.72	4.76	9.00	44.72
B8 before	54.17	7.70	14.30	55.28
BI8 after	51.39	7.47	13.70	52.50
KD4 before	51.39	7.26	13.40	51.94
KD4 after	48.61	6.65	12.40	49.17
PM7 before	58.61	9.20	16.70	60.28
PM7 after	58.06	8.16	15.10	59.17
SI10 before	54.72	9.49	17.60	55.83
SI10 after	55.83	9.67	18.00	56.94

CONVERSATION

Subjects	DAx1 mean	SD DAx1	DAx1 Range	DAx2 mean
SB5 before	53.06	7.98	14.90	54.17
SB5 after	45.28	5.48	10.80	45.28
BI8 before	53.06	10.08	18.90	54.17
BI8 after	51.39	8.66	16.30	52.50
KD4 before	48.61	8.37	15.60	49.17
KD4 after	50.83	7.80	14.40	51.39
PM7 before	55.83	9.87	18.30	57.50
PM7 after	57.50	10.10	18.30	58.61
SI10 before	53.61	9.56	17.80	54.72
SI10 after	52.50	9.45	17.90	54.17

Appendix R

Statistical significance between the Parkinsonian group before and after medication in reading compared to conversation

Within Subjects ANOVA[16]

Variable	Parkinsonian (before medication) *p*	Parkinsonian (after medication) *p*	Overall effect *p*
DFx1 mean	0.176	0.273	-
SDDFx1	**0.001**	**0.001**	**0.000**
DFx1 90% range	**0.018**	**0.018**	-
DFx2 mean	0.140	0.166	0.119
SDDFx2	**0.017**	**0.020**	-
DFx2 90% range	**0.001**	**0.002**	**0.001**
DAx1 mean	**0.001**	0.340	**0.001**
SDDAx1	1.00	1.00	-
DAx1 90% range	**0.012**	**0.004**	**0.005**
DAx2 mean	**0.001**	0.364	**0.002**
SDDAx2	1.00	1.00	-
DAx2 90% range	**0.002**	**0.004**	**0.001**
DQx1 mean	0.876	0.403	0.698
SDDQx1	**0.022**	**0.012**	**0.003**
DQx1 90% range	**0.017**	**0.022**	**0.002**

[16] These results were confirmed with non parametrics (Wilcoxon) that showed the same statistical differences. There were 3 cases that no normality of distributions was found (DFx1 mean, DFx1 90% range, SDDFx2). In these cases Wilcoxon paired samples test was used.